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Original Communications

THE INFERIOR ESOPHAGEAL CONSTRICTOR IN RELATION TO LOWER ESOPHAGEAL DISEASE

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IT IS interesting to note the lack of definite information and the conflicting opinions relative to the anatomy and physiology of the esophagus and to the pathogenesis of esophageal diseases such as the Plummer-Vinson syndrome, esophagospasm, achalasia or mega-esophagus, pulsion diverticula, and idiopathic strictures. This lack of knowledge results from the inability either to produce such conditions experimentally or at least to correlate experimental and clinical findings. It is also difficult or impossible to deduce the genesis of pathologic conditions from gross and microscopic findings of well-established chronic lesions. It is also difficult or impossible to determine whether such findings may be primary and part of the etiology or entirely secondary manifestations. In view of this situation observations relative to these conditions should be presented by those interested in this field in an attempt to help clarify our understanding and perhaps help as an eventual aid in therapy.

The esophagus has a well-developed outer longitudinal muscle layer and a less well-developed inner muscle layer. The longitudinal muscle extends from the cricoid cartilage and cricopharyngeus muscle above to insert below in an interlacing manner into the middle and outer muscle layers of the cardia. The inner muscle layer commences in the upper end independently of other groups of muscle fibers. The arrangement of these fibers varies in the different segments of the esophagus. They run in an elliptical direction around both sides of the esophagus and downward in the upper third; they are circular for a small segment in the middle third, below which they again run in an elliptical course in a downward direction toward the posterior aspect.

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result of reduction or degeneration of Auerbach's plexus noted in many patients with mega-esophagus. The sphincterlike action in the terminal esophagus of patients with mega-esophagus can be demonstrated fluoroscopically to dilate under the influence of amyl nitrite and ergotamine. The dilating action of amyl nitrite on the sphincterlike mechanism of the hypertrophied circular muscle in the terminal esophagus has also been observed in some cases of achalasia at the time of surgical dissection.[5]



Fig. 1.—*A*, Esophagram of a patient with achalasia of the esophagus demonstrating the point of obstruction approximately 1 inch above the cardia at the site of the inferior esophageal constrictor. *B*, Surgical exposure of achalasia of esophagus demonstrating the site of inferior esophageal constrictor in its contracted state. *C*, Surgical dissection of the inferior esophageal constrictor.

Esophageal obstruction will cause secondary hypertrophy of both the longitudinal and inner muscle layer above the point of obstruction. The hypertrophy of the circular layer of the so-called inferior esophageal constrictor in patients with mega-esophagus far outproportions hypertrophy found elsewhere. The hypertrophied muscular constrictor or sphincter has been found in all fifteen patients with achalasia operated upon in the last two years during which time a search for this constrictor was made and a myotomy performed. Prior to this time a Heineke-Mikulicz or Finney cardioplasty was



The etiology of pulsion diverticula of the lower thoracic esophagus has never been satisfactorily given. Most authors have alleged that there is a congenital weakness of the esophageal musculature or that there is a weakness



A.

B.

Fig. 3.—*A*, Surgical exposure of a pulsion diverticulum. *B*, Arrow points to the transected hypertrophied circular muscle fibers of the inferior esophageal constrictor just distal to the stoma of the pulsion diverticulum.



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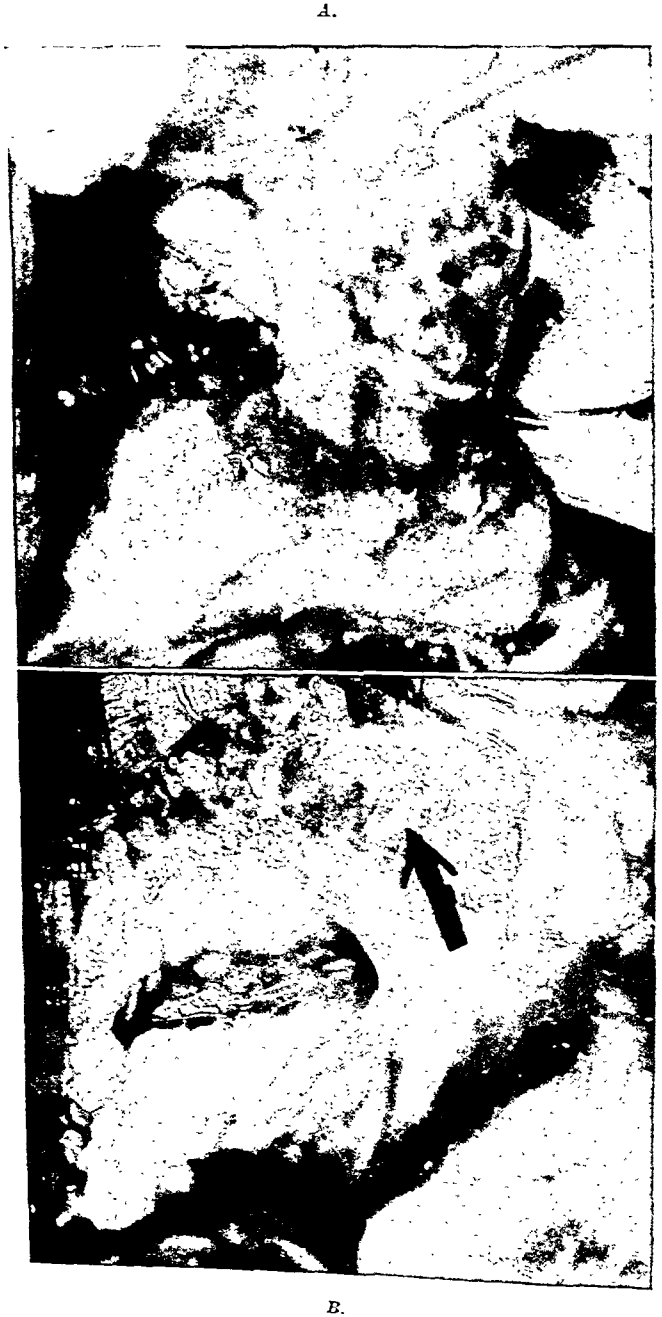
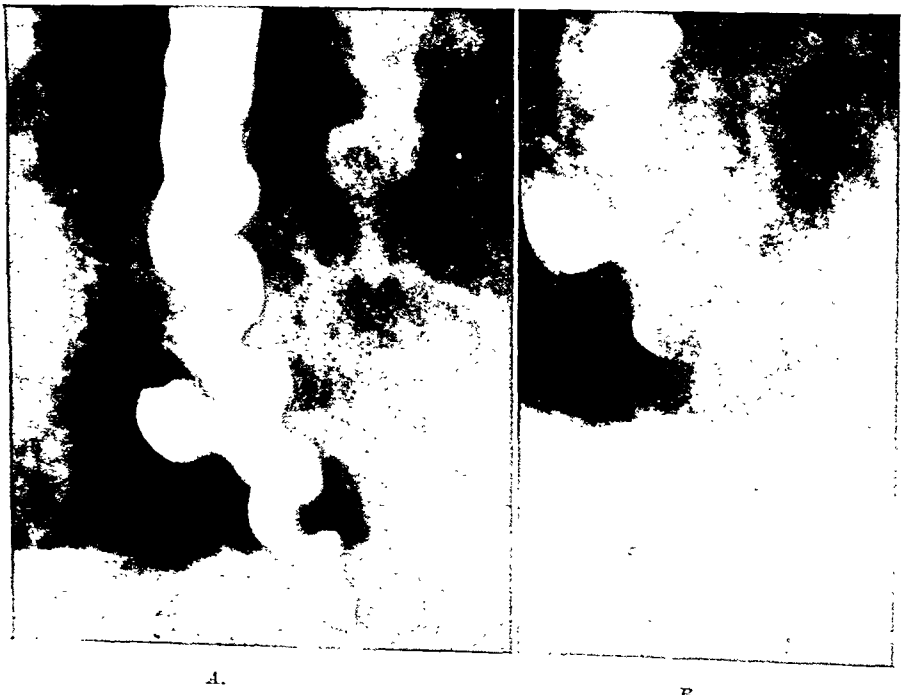


Fig. 3.—*A*, Surgical exposure of a pulsion diverticulum. *B*, Arrow points to the transected hypertrophied circular muscle fibers of the inferior esophageal constrictor just distal to the stoma of the pulsion diverticulum.

muscle; whereas, above the stenosis produced by the carcinoma there was generalized hypertrophy of both the circular and longitudinal layers to the same degree. The carcinomas in these cases were probably secondary to chronic irritation due to the diverticula. Microscopic examination of nerve fibers and the ganglion cells of Auerbach's plexus failed to show evidence of nerve degeneration in these two patients.

In the last case, a 77-year-old man with a history of dysphagia of twenty years duration, evidence of mega-esophagus and a pulsion diverticulum were both present. In addition to this there was a bilateral chronic pneumonitis due to many years of regurgitation and bronchial aspiration. Swallowing had



A. *B.*

Fig. 4, *A* and *B*.—Esophagrams demonstrating functional stenosis at the site of the inferior esophageal constrictor, tertiary contractions of the esophageal musculature, and a pulsion diverticulum.

become almost entirely impossible for a period of two months. An esophagram obtained fourteen years previously at another hospital demonstrated the presence of both a large pulsion diverticulum and a mega-esophagus (Fig. 6, *A*). Esophagrams obtained prior to surgery (Fig. 6, *B*) demonstrated the diverticulum to be so large as to overshadow completely the mega-esophagus radiographically but the markedly dilated esophagus was apparent fluoroscopically.

At the time of the operative dissection (Fig. 7, *A*) a markedly hypertrophied circular muscle band was noted just distal to the stoma of the diverticulum which appeared to be acting as an obstructing agent. Dilatation and tortuosity of the esophagus were present mainly above the stoma of the diverticulum. Because of the esophageal redundancy and dilatation above the



at home in order for him to gain weight and strength. After four days of this vigorous feeding policy he suddenly developed pain in the right chest and was readmitted to the hospital. He was found to have a right empyema secondary to esophageal perforation. He soon developed cardiac failure and expired. Post-mortem examination of the esophagus revealed a perforation of the suture line. Just distal to the perforation the inner circular muscle was found to be considerably more hypertrophied than that upon the proximal side as demonstrated in Fig. 7, *B*. It was felt that the constricting element of the hypertrophied inner circular muscle in the lower esophagus responsible for both the achalasia and the pulsion diverticulum had been incompletely removed at the time of the esophagectomy and was still causing sufficient functional stenosis to be responsible for the subsequent perforation at the anastomosis.



A *B*.

Fig. 6.—*A*, Esophagram demonstrating an achalasia of the esophagus with dilatation except in the terminal esophagus and a large pulsion diverticulum. *B*, Esophagram of the same patient fourteen years later demonstrating increase in size of pulsion diverticulum.

In view of these observations it is felt that functional stenosis in the distal esophagus from a hypertrophied inferior esophageal constrictor causes increased intraesophageal pressure and herniation of the mucous membrane through weak areas in the inner muscle layer to form pulsion diverticula. The cause of the hypertrophied inferior esophageal constrictor is still problematical but probably related to one of the theories predisposing to a preponderance of sympathetic control as discussed previously in this paper.

The next and last group that may have a similar etiology are the strictures in the distal esophagus occurring in the older age group. Three types of patients may or may not fall into this group. The first group includes those patients with hiatal hernias who may have a relaxed or hypotonic inferior

A.





esophageal constrictor associated with a relaxed or atrophic diaphragmaticoesophageal ligament. The ascending limb of this ligament arises from the fascia on the undersurface of the diaphragm, extends through the esophageal hiatus, and attaches itself to the longitudinal and circular muscle fibers of the lower esophagus at the site of the inferior esophageal constrictor or sphincter. This ligament is the antagonist of the longitudinal muscle of the esophagus and prevents the esophagus from pulling the stomach into the chest during swallowing.



A. *B.*

Fig. 8.—*A*, Esophagram demonstrating an esophageal stricture secondary to a hiatal hernia. The atrophic diaphragmaticoesophageal ligament allows the stomach to herniate into the mediastinum and a hypotonic inferior esophageal contractor allows regurgitation of gastric juice with secondary peptic esophagitis. *B*, Esophagram following treatment by bouginage and ulcer management.

There appears to be an interrelationship between the diaphragmaticoesophageal ligament and the inferior esophageal constrictor in reference to the opening-closing mechanism of the terminal esophagus. Normal individuals can obstruct the flow of barium in the terminal esophagus by taking a deep inspiration at which time the ascending limb of the diaphragmaticoesophageal ligament becomes taut. This can be demonstrated to a greater degree in patients with achalasia. Furthermore the inferior esophageal sphincter in patients with achalasia appears to be hypertrophied.

Opposed to the findings present in achalasia, the relaxed or atrophic diaphragmaticoesophageal ligament present in hiatal hernias allows the stomach to herniate into the posterior mediastinum, and the hypotonic in-

ferior esophageal constrictor allows regurgitation of gastric juice with secondary peptic esophagitis (Fig. 8). If the esophagitis persists, secondary scarring and stricture formation in the terminal esophagus develop. Furthermore, permanent shortening of the esophagus may occur with loss of the sliding action of the hiatal hernia. This syndrome is frequently referred to as a congenital short esophagus with thoracic stomach. The latter condition does occur but its incidence is not as great.



A. B.

development of the stricture. Esophagitis can frequently be seen by esophagoscopic examination.

The third type includes those patients who develop difficulty in swallowing and who do not have pre-existent esophageal or gastric complaints. These patients are usually older and have evidence of other degenerative diseases. In such patients it is possible that the organic stricture may be secondary to esophagitis produced by stasis resulting from functional obstruction by a hypertrophied inferior esophageal constrictor (Fig. 9). Most of these patients can be relieved by medical management including an ulcer diet, other buffers, and esophageal dilatation so that further observations from surgical dissections are lacking. The periesophageal fibrosis and mural scarring present in those patients requiring surgical resection of the stricture mask any possible etiological factors.

SUMMARY

Observations are presented which suggest that either spasm or hypertrophy of the inferior esophageal constrictor may cause functional stenosis and be a factor in the production of functional dysphagia, the Plummer-Vinson syndrome, esophagospasm, achalasia, pulsion diverticula of the thoracic esophagus, and idiopathic strictures of the terminal esophagus. The etiology of this spasm or hypertrophy is not clear but undoubtedly is similar to spasm of circular muscle in other smooth muscle sphincters in the body. There is both experimental and clinical evidence to suggest that this spasm and hypertrophy result from a preponderance of sympathetic control. Possible causation of this sympathetic imbalance is discussed.

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DISCUSSION

DR. I. DARIN PUPPEL, Columbus, Ohio.—I think we should all congratulate Dr. Kay for his contribution to the understanding of achalasia and other diseases of the lower end of the esophagus.

Certainly there is still much lacking in our knowledge of the etiology and the treatment of this group of diseases, particularly achalasia. We know that normally esophageal swallowing depends to a great extent upon peristaltic movements which expel the intraluminal contents of the esophagus into the stomach. At least those of us who have lived on farms know that in childhood we were able to drink uphill from a spring of water. This upward esophageal swallowing was dependent almost entirely upon peristaltic movement of the esophageal musculature. Hurst, in his important studies and papers over a period of about two decades in the early part of this century, emphasized that the achalasia disease was

localized to the cardiac end of the esophagus. It was common opinion that peristalsis occurred normally in mega-esophagus but that it was ineffective in deglutition because the bolus of food was not large enough to be grasped by the hugely dilated esophagus. However, in recent years, many investigators have been convinced that this disease is much more generalized, involving much more of the esophagus than was heretofore realized. We, for instance, published a paper in the JOURNAL OF THORACIC SURGERY a few years ago, in which we emphasized that in four cases of achalasia there was a definite involvement of the whole esophagus in functional disturbances. Not only the smooth but also the striated musculature of the esophagus was involved in these disturbances. These physiologic abnormalities were not necessarily greatly changed by relief of the obstruction at the cardia. Using inflated balloons at various levels of the esophagus which acted as boluses of food to stimulate the esophagus to movement, and recording the movements on a long kymograph, we found the intrinsic motility of the whole neuromuscular esophagus in achalasia to be irregular, uncoordinated, hyporesponsive to stimuli, and never completely effective. In fact, the intrinsic motility was completely absent in some cases. Kramer and Inglefinger, of Boston, are the only men to our knowledge who have done similar studies. It was interesting that they, publishing their work almost one year after we had submitted our paper for publication in the JOURNAL, independently confirmed that portion of our research in which they were interested.

We now have similarly studied eight additional cases of achalasia. We know from these experiences that every so-called idiopathic mega-esophagus shows a profound change of varying degree of the intrinsic motility with little or no production of peristalsis and great decrease in the power of deglutition. Usually in the fusiform type we found slight disturbances, in the flask type moderate disturbances, and in the sigmoid or zig-zag type, which we feel is the far-advanced type of mega-esophagus, we have found none or little intrinsic motility. Barrett has recently written that he has never seen the fusiform transform into the sigmoid type of mega-esophagus. Yet I have available roentgenograms of barium swallow studies of one patient over a period of fourteen years. These revealed a fusiform mega-esophagus in 1937 which transformed to a sigmoid type by 1951. I did an esophagogastrostomy, bilateral vagotomy, and a posterior gastrojejunostomy for this patient and she is now well.

In two cases of uncomplicated supradiaphragmatic diverticula of the esophagus, as proved on excision at surgery, we were not able to elicit preoperatively or postoperatively disturbances of the esophagus proximal to the diverticulum. We feel that the two diseases, physiopathologically speaking, are different in nature.

When we administered a sympathicolytic drug such as Priscoline or a parasympathomimetic drug such as Urecoline alone or in combination, we found that the mega-esophagus lumen usually reduced in size and some increase in the rate of emptying of the esophageal contents occurred. Tracing studies showed frequently marked increase in tonus, frequency, and power of contractures of the esophageal musculature. And yet these drugs were disappointing in their therapeutic value for the reason that almost always the vigorous movements still remained localized, pathologic, uncoordinated, and nonperistaltic in nature. They merely led to an increased kneading or churning effect so that the power of esophageal deglutition was usually not appreciably enhanced, thus differing from the effects of these drugs on the hypomotility of the atropinized normal esophagus.

Our data indicate there is a marked disturbance of the physiology of the whole esophageal neuromusculature. They indicate that our present modes of treatment cannot entirely relieve the abnormal physiology and that other better concepts will have to be sought. All present methods of treatment have their disadvantages and failures. We now routinely do preoperative overnight twelve-hour gastric secretion and histamine gastric analysis studies. For those patients who show free hydrochloric acid we have used Banthine, and have done bilateral vagotomy together with a gastrojejunostomy or a partial gastric sympathectomy besides relieving the esophagogastric obstruction, and have noted remarkable improvement in end results in certain cases. Esophagitis did not occur in two cases maintained achlorhydric postoperatively. Achalasia of the esophagus is a progressive disease and, on the whole, better therapeutic end results may be expected if the diagnosis is made early and treatment is instituted early in the course of this disease.

DR. J. KARL POPPE, Portland, Ore.—I would like to take this opportunity to mention one method of treating epiphrenic diverticula which should not be used. I refer to my own experience with two cases of large epiphrenic diverticula associated with esophagitis, diverticulitis, stricture, and spasm of the lower esophagus and cardia. In each of these two patients a very simple method was used, anastomosing the fundus of the stomach to the bottom of the diverticulum, a relatively simple procedure which can be done readily, relieving the diverticulitis. This anastomosis worked well for a period of approximately eight months in one case, at which time the suture line apparently opened and a fistula occurred between the diverticulum and the pericardial sac. We drained that by a large pericardiotomy and the food came out through the pericardiotomy opening in the anterior chest for a few weeks. The fistula gradually closed by an obliterative pericarditis. The patient unfortunately expired from an infarction in the right lung a few weeks later.

The other patient did much better than that. Swallowing was satisfactory for about three years, at which time he also developed a fistula and began to cough up food. I understand this required a lobectomy and resection of the lower esophagus. A radical resection of the lower esophagus including the diverticulum and stricture should have been done in the first place to remove the infected diverticulum and obstruction of the lower esophagus in each case.

DR. J. L. EHRENHAFT, Iowa City.—We have recently been interested in this particular problem, and at the present time there are functional motion studies being carried on by Dr. Taber in our hospital which are rather interesting. Up to the present time all the studies have been done by single balloons placed in the esophagus. Dr. Taber has inserted three balloons at different levels, hypopharynx, mid-esophagus, and lower esophagus, which are swallowed, and they are connected simultaneously to very sensitive kymographs. We have studied about six or seven patients with achalasia up to the present time. This being a functional disease which is certainly progressive, we see these patients quite late. The tone and the peristalsis of the muscles are greatly decreased. We recently had a patient who was being treated for another condition, whom we used as a control for a normal esophagus. At the time the balloons were swallowed and the motion studies were done, there was complete absence of contraction of the esophageal musculature. Esophagrams showed a dilated esophagus with cardiospasm.

EXPERIENCES WITH THE OPERATIVE MANAGEMENT OF DELAYED RESTORATION OF ALIMENTARY CONTINUITY IN CHILDREN ORIGINALLY TREATED BY THE MULTIPLE STAGE PROCEDURE FOR CORRECTION OF CONGENITAL TRACHEOESOPHAGEAL DEFECTS

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INTRODUCTION

Among most surgeons who deal with the congenital tracheoesophageal fistula problem there currently exists a clearly understandable sentiment for salvaging the greatest possible number of children. To those deriving guidance from this principle, it is apparent that the routine insistence on the use of a primary anastomosis will inevitably yield a smaller percentage of successes. Contrariwise, an opportune use of the multiple stage procedure, in the instances of simple atresia, will often save the life of one more child. Regardless of one's scientific or emotional bent, and we personally strongly oppose an "all-or-none" operative philosophy for the management of this grave ailment, an occasional need will surely arise for techniques of bridging a substantial segment of the esophagus. Because of these considerations we are reporting our experiences with the resolution of this situation in ten children originally treated by the multiple procedure technique of fistula ligation, cervical esophagostomy, and gastrostomy.

HISTORICAL

In an earlier presentation with Lannin and Tongen[12] we reviewed the items of the embryologic variety of lesions occurring in congenital atresia of the esophagus, the historical attempts at therapy for this problem, and our experiences with 103 consecutive cases,* and considered in some detail the general and the specific surgical care for this situation. From this group, there were ten children originally treated by the multiple stage procedure, on whom we operated for restoration of alimentary continuity.

During the years intervening since Leven and, independently, Ladd achieved success with the multiple stage technique, numerous surgeons have skirmished with this problem in reconstruction. Some have sought to develop an antethoracic skin tube. Ladd,[8] Ivy,[7] and Lam[11] did employ some variant of this plan for completing intestinal continuity in the tracheoesophageal fistula cases; Davis and Stafford,[3] Stevenson,[21] and Hanrahan[5] developed skin-lined simulated gullets for comparable situations. Though completed tunnels were

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*This includes sixty-eight instances of primary anastomosis with forty-three survivors.

reported, early and late technical tribulations were met. Closure difficulties arose from primary or recurrent fistula formation as well as from strictures eventuating particularly at the gastrocutaneous anastomotic junction. Ochsner and Owens,[16] Lam,[10] and Ladd and Swenson[9] sought to reduce the item of scarring from acid peptic juice by bringing to the anterior abdominal wall the distal esophageal segment.

The effectiveness of this maneuver in reflux control is questionable, however, since the dissection required would seriously compromise one's normal esophagogastric pinchcock mechanism. These several limitations contributed to a rather tepid enthusiasm among many surgeons for this general management program and did encourage the planning of alternative approaches.

Yudin[25] has described in some detail many techniques, essentially of historical interest and but infrequently attempted currently save for that procedure pioneered by César Roux. The use of a limb of jejunum, secured from the segment just distal to the Treitz ligament, as an antethoracic esophageal replacement, received the major attention of Yudin. In fact, the Y-type anastomosis seemed to represent his preferred method for bridging long caustic or acid-induced esophageal strictures. His description and suggestions for the technical management of the mesenteric dissection served reliably for guidance while we, from a quite inexperienced position, were acquiring some familiarity with this problem. A careful study of this paper is recommended for those planning to use the jejunum for thoracic esophageal replacement. Despite Yudin's favorable support of the method, the development of a length of vascular mesentery so adequate and uncompromised for viability as to permit a safe union with the cervical esophagostomy was at times a poser and led to its virtual abandonment by some.[4] A clever and singular success in meeting this issue was described by Longmire[14] who reanastomosed one set of the vasa recta jejunalis to the internal mammary vessels. Longmire and Ravitch[13] effectively developed a parasitic blood supply from the surrounding pedicle tube for a length of transposed jejunum by a careful staging through several steps so that ultimately complete division of the mesenteric tributaries was achieved.

Van Prohaska and Sloan[23] sought to realize a greater transplantable length in the jejunal segment by denuding the mucosa of the serosal and muscular layers. The remaining free, full thickness, mucosal tubes were rather uniformly nonviable in their canine experiments, and use of this technique was followed by some stenosis in one clinical counterpart. Harrison,[6] as well as Robertson and Sarjeant,[19] shortened the distance to be bridged by recourse to a more direct routing of the jejunal limb between cervical gullet and stomach. Rather than the usual arclike position described by a subcutaneously placed antethoracic intestinal segment, the bowel was carried more like a secant directly through the thorax by the former and just substernally by the latter team. Cosmetically, these methods revealed certain advantages, as well as minimizing any likelihood of complications secondary to severe compressive trauma against the firm chest wall. On the other hand, to all persons cognizant of children's dietary indiscretions, the accidental impaction of some unusual sized particulate bolus within

this intestinal segment is a possibility and might likely be at a quite inaccessible site. Endoscopic instrumentation through the kinked bowel segments, too, would be hazardous.*

Rienhoff,[18] Bigger,[1] and Potts[17] have mobilized the stomach to complete a delayed antethoracic esophagogastric anastomosis. The latter author subsequently placed these segments within the left thoracic space after division of the appropriate costal cartilages.

Sweet[22] and Clifton,[2] in a delayed reconstruction of alimentary discontinuity secondary to esophageal atresia, moved the stomach into the chest and out the superior thoracic aperture. A gastric anastomosis with the cervical esophagus was completed in two cases recorded by the former. Food consumption was described as essentially normal, though growth came a bit slowly to one child among Sweet's cases. During subsequent years this child improved at a more rapid pace. No untoward physiologic disturbances were noted or ascribable to the intrathoracic stomach. In a subsequent publication, Longmire[15] carried through a reconstruction plan resembling Yudin's, in that a jejunal segment pedicled on a single vertical mesenteric tributary was mobilized into an antethoracic position. By this technique, alimentary continuity was restored to two infants shortly after thoracic exploration revealed the nonanastomosable hypoplastic lower esophageal segment in association with a tracheoesophageal fistula. Their subsequent physical development and health as recorded in a follow-up at twenty-eight and thirty-nine months, respectively, has been gratifying. Delayed sectioning of alternate principal elements of the vascular rete has been studied by Shumacker.[20] For the rat and for the dog, by this means, a greater length of intestine could safely be pedicled. Its successful application to a clinical case of lye stricture in a child was described.

Our own first cautious groping for some acceptable solution to this situation began in 1947. For minimum standards we desired a technique surgically safe, compatible with a reasonable rate of physical development, and one which would be unlikely to prejudice the future should an anatomic rearrangement at a later date prove essential. It should also have enough cosmetic appeal to prevent rejection or scornful comparisons when this child would be exposed to the comparative glances and comments of his playmates. The reconstructions in nine of the ten cases being reported seem to meet these requirements reasonably well.

METHOD

As a general plan these children were admitted a few days before the first stage of the reconstructive procedure. During these twenty-four hours immediately prior to surgery, at least two intramuscular penicillin injections were given; streptomycin or Aureomycin was supplied via the gastrostomy, but no nutrient was permitted by this route during the last twelve preoperative hours. The anesthetic for each stage in virtually every operation was Pentothal Sodium and curare, for induction and for maintenance. A high concentration of oxygen in combination with nitrous oxide was supplied through an endotracheal tube. These anesthesias, administered by a large and changing number of the medical personnel in training for that specialty, were invariably superbly well done. Midline, right or left upper

*In one of our cases, a swallowed penny became so inextricably wedged into the esophageal pouch as to require transcervical removal.

rectus, or transverse incisions all offered an adequate exposure. The incision of preference of the original operator at the time the abdomen had been entered to make the gastrostomy was reopened to develop the exposure about the area of the jejunal mesentery. Any and all adhesions to the upper small bowel and its mesentery were first freed. The sheaths of peritoneum were preserved over all arcuate vessels immediately next to the bowel. It was cleared away, however, along with all fat and often a considerable quantity of variously sized and clustered lymph nodes, directly down to the first portion of the main superior mesenteric vessel to the entire small bowel. This arterial segment and the first four or five major primary channels were carefully dissected free of the network of sympathetic fibers ramifying locally and distally. The vasa recta jejunalis to be responsible for nourishing the transplant and the superior mesenteric artery at this level was denuded of autonomic fibers and areolar substance quite as carefully as one prepares a small artery for an anastomosis. Vasospasm was not sustained in these periarteriectomized segments. With serrefine clamps applied to as many as three or four of these main tributaries, reflections from pulsations were usually visible along the vascular arcades immediately adjacent to the moist bowel. The first large vascular channel to the jejunum was always saved to insure a sound blood supply on the proximal line of the intestinal anastomosis. With the bowel sectioned 6 to 10 cm. distal to the Treitz ligament it as periodically tested for adequacy of length by measuring it onto the anterior abdominal and chest wall. Redivision of the straight vessels and then occasionally those of the first arcade served to straighten the intestinal length relative to mesenteric length. After experiences with uniting the upper end of this jejunal segment to the esophagus, once or twice, it was self-evident that enough redundant bowel to bridge any reasonable gap could be dissected free at that time. Any all out effort to bring this jejunal limb precisely to the cervical stoma was therefore unnecessary and might even compromise its terminal few centimeters. After developing an approximately adequate length of this prepared portion of bowel, it was passed through a rent in the transverse colonic mesentery and then brought out from the abdomen in a midline fascial opening. It then dipped into a widely undercut subcutaneous tunnel, finally popping out through a loosely fitting skin incision at about the level of the clavicle or first rib. To minimize tension in this antethoracic tunnel the area of the chest and abdominal wall was widely freed over an area from the right side of the sternum to the left anterior axillary line. Serosanguineous accumulations in this raw region were led away by two or three laterally placed soft Penrose type rubber drains coming out of stab incisions. Intestinal continuity was re-established by end-to-side union (Roux Y) below (retrocolic) the transverse colonic mesentery. This tissue was tacked to the loop of jejunum and then the abdominal fascia was sewn to the bowel as it left the peritoneal cavity—quite similar to the technique for preventing herniation about an ileostomy. Attention was ever directed against the development of mesenteric venous engorgement in the transplanted loop either through compression or twisting, or from inadvertently placed sutures. The short external segment of bowel was left open to preclude the serious accumulation of internal tension from secretions. It usually became cyanotic in varying degrees. Its viability was tested and deemed all right in the first patient by fluorescence to ultraviolet light after an intravenous injection of fluorescein. Continuity in the cervical region was accomplished at intervals of a few days to several months following the development of the antethoracic jejunal segment. In the first cases, this delay was deliberate in order to permit the edema and other acute inflammatory reactions, aroused by the first procedure, to subside, so that the bowel could then be more easily sewn to the esophagus. The likelihood of a salivary fistula of even a few days' duration is probably lessened by a reasonable exhibition of patience. These anastomoses were all of the open type, two rows, with the inner one mucosa-to-mucosa union, and were completed with 00000 silk.

Once we recognized the essential need for correcting the "by-pass" of the stomach by the jejunal segment, another stage was added to the reconstruction. The abdomen was opened once more and the jejunal limb of the Y anastomosis divided close to the duodenum, leaving a small blind pocket. The excess bowel was now resected after devascularizing it close to the mesenteric border. Several kinks thereby could usually be removed. This straightened segment was then joined to the antrum. Most of the feeding gastrostomies were

maintained for several months to supplement the child's caloric intake. When eventually abandoned they rarely healed spontaneously, but required a coring out of the tract plus a formal closure of that area.

In one child, J. N., intestinal continuity was established by transplanting the entire stomach into the left thorax. This dissection was carried out through a thoracoabdominal incision. An esophagogastric anastomosis was made in the neck after an extraperiosteal removal of the first rib provided an ample-sized opening to deliver into the cervical area the gastric fundus.

RESULTS AND DISCUSSION

To date the reconstructions have been completed in eight children through the stages of esophagojejunostomy, jejunogastrostomy, and closure of the gastrostomy. Their ages, current heights, weights, and dates of last operation are listed in Table I. One boy has only the cervical esophagus anastomosed to the jejunal limb; another child has had alimentary continuity established by esophagogastrostomy after transplantation of the stomach into the left thorax and lower neck. The first reconstruction was started as a jejunal transplant in a 3½-year-old boy on June 5, 1947. On Aug. 21, 1947, the upper stoma was united to the esophagostomy and convalescence was uneventful. At times, and unpredictably in relation to the kind of meal eaten, this child has had some of the manifestations of a "dumping" syndrome, that is, tachycardia, sweating, and pallor. He would often then express a desire to lie down. Nausea was not noticeable. There was a slow but steady gain in weight and appetite evidenced by the youngster. Because of his generally acceptable over-all status the need for transplanting the jejunum back onto the stomach was not at once apparent.

TABLE I. POSTRECONSTRUCTION GROWTH RECORD FOR ALL CHILDREN AS OF MAY 1, 1952

PATIENT	AGE (YR.)	CURRENT WEIGHT (POUNDS)	CURRENT HEIGHT (INCHES)	DATE RECONSTRUCTION COMPLETED
D. E.	9			4/29/49
D. W.	8½	55	51	9/ 9/49
D. G.	9	66¼	54½	10/25/49
O. B.	6½	51½	47	11/15/49
J. B.	8	51½	46½	12/30/49
J. N.	7	33¾	45	1/20/50
R. L.	12	80	56½	3/31/51
G. B.	6½	35	43	6/ 6/51
M. S.	5	37	42	7/24/51
D. S.	5	40½	43	9/28/51

Our second patient, anastomosed for oral alimentation Sept. 16, 1948, did so poorly in the following few months (alopecia areata, little weight gain, reluctance to eat, and frank personality changes) that a jejunogastrostomy was performed in April, 1949. Subsequent to this procedure the child improved steadily and he has maintained a fair growth rate since. In other cases of this series we have noted the progressive improvement in the individual's general health once the stomach has been restored to the direct line of alimentary continuity. Longmire has recently commented upon the importance of not "by-passing" this organ. (Yudin's statement to the contrary might be accounted for by the older age of the patients in his series, and hence their lowered

metabolic rate.) For children, we believe there are real advantages to having it connected directly into the food circuit. The antrum was selected in all cases for union with the jejunum. The anastomosis was deliberately placed here recognizing that should a stomal ulcer ever trouble the individual later in life, it would still be possible to resect, transthoracically, enough of the acid-bearing area to cure the ulcer diathesis, without disturbing those already existing anastomoses. Theoretically and from experimental evidence there is a somewhat greater likelihood of an ulceration occurring in the situation where the stomach is "by-passed" than if continuity is made with the jejunum.

The present weight and height of each child in this group have been plotted on a Wetzel grid.[24] Seven of the nine individuals in this group cared for by the jejunal transplant were above or around the 67 per cent auxodrome with three of these sustaining physiques and developmental levels to the 15 per cent auxodrome. When these items of weight, height, and age were plotted for the child reconstructed by an esophagogastrostomy technique, the curve derived was barely on the grid, and far below the 98 per cent auxodrome. In the twenty-seven elapsed months since completion of his anastomosis, this youngster has remained fragile and "puny." Therefore, the likelihood is strong, particularly since he has fallen away from his isodevelopmental line, that this case represents one with a poor physical fitness record. Clinically, there is support for this type of representation. Whereas eating seems to provoke in him no acknowledged distress, exposure to and selection from a variety of nutritious items at any meal rarely captivates his appetite. Roentgenologically the stomach appears flaccid, empties slowly, but ultimately, and shows no organic site of obstruction. It is conceivable that, for this child, vagotomy with its secondary inhibitory effects on pancreatic function has been majorly responsible for a substantial measure of the obvious physical retardation. Obversely, most of the children with a jejunal reconstruction including an anastomosis onto the stomach have done well, and several have sustained an excellent physical fitness record.

The youngest child in this series was 2 years, 9 months of age at the time a jejunal limb was positioned onto the chest wall. The remainder were several months to years older before a surgical reconstruction was attempted. This uncertainty and admittedly timorous viewpoint about beginning the necessary reparative steps on very young children was fostered by an often expressed desire from the parents to await a "best" operation. Several were participants in a close network of correspondents composed of parents raising similarly crippled children. Longmire's excellent results in restoration of oral alimentation with two neonatal infants offer a sound argument in favor of an earlier intervention than we practiced.

Although these youngsters uniformly came to the initial operation for restoration of alimentary continuity in good physical condition, the burden of preparing and patiently injecting thousands of gastrostomy feedings, and then diapering the cervical esophagostomy, was assuredly an enormous labor of love. In this regard some parents had been farsighted enough to encourage their

child to eat and drink from a wide selection of foodstuffs and beverages. This painstaking cultivation of youthful taste buds and palate was rewarded by a more eager acceptance of regular childhood fare once intestinal continuity was achieved. The adjustment, in mouths tender from no contact with the coarser foods, was slower.

The cosmetic result is not nearly perfect in any single case, at least to our critical gaze. Fortunately, the parents have been more charitable, and realistic. There has been no ardent acceptance of our offer to improve the child's appearance by placing the at times prominent intestinal loops beneath the thoracic frame. Of course, this suggestion has always been coupled with an explanation that this procedure could do nothing to improve intestinal function. We have also speculated about the hazard from trauma to the bowel or to its blood supply, by a crushing injury applied to the chest or costal margin. Specifically in boys this potential has provoked some concern for the future. True, the follow-up time has been short, and these children with but a single exception are rather too young for the hardier athletic contests. Still, for this oldest boy, roughhousing, wrestling, and back yard football of a somewhat rugged character have provoked no distressing symptoms nor daunted his zest for such exercise. Some parents are yet considering this final transplantation plan, but most of them are outspokenly grateful now that their son or daughter can eat contently and from the family table. With a greater experience in treating the arcades and vertical nutrient vessels, straighter, less redundant, segments of interposed jejunum have been left. Also, the excess intestine can be tailored anew and shortened at the time of completing the gastrojejunal union. This is one small argument in favor of delaying that stage until a later date in the over-all restoration program. As noted often in the whole series, during fluoroscopic examination of this segment, the main bolus of barium drops under gravity's influence and is pushed by peristalsis through to the gastric pouch, seconds after its oral ingestion. An inability to empty this intestinal limb, granted the meal is not a particularly dry one, has not arisen as a handicap to the acceptance of a good-sized repast in the course of one sitting. A small serous or salivary fistula was noted at the upper anastomosis in three cases. All healing was spontaneous, with the child remaining on clear liquids, in the course of three to five days. The remainder of the cervical esophagojejunal junctions healed primarily without evidence of gross leak, as did all of the intra-abdominal suture lines. In the tenth and last patient operated upon, a slough developed at the superior end about twenty-four hours after the surgery for the first stage. Prior to this time the color in this segment had seemed quite satisfactory. Promptly after the necrotic end was recognized the subcutaneous tunnel was opened under local anesthesia at mid-chest and all nonviable or dubiously viable bowel cut free. Fortunately, an adequate length yet existed to permit approximation of the remaining segment to the cervical esophagostomy at a later date. In this boy the jejunal limb is now straighter than it is in some of the other children, and it functions smoothly.

SUMMARY

Experiences with ten patients in the operative management of delayed restoration of alimentary continuity in children originally treated by the multiple stage procedure for congenital tracheoesophageal defects have been reported. The shortcomings of both methods tried, as well as their advantages, have been noted. The functional results in those cared for by the interposition of a jejunal limb between the cervical esophagus and the gastric pouch were appraised as superior. The single child with stomach united to the upper gullet has failed to mature physically at a standard and acceptable pace. The relevancy and need for a sustained follow-up in this group and in comparable cases, through their later formative years to test the effect of these stresses, as well as aging, are candidly conceded.

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DISCUSSION

DR. FREDERICK G. KERGIN, Toronto.—I am very interested in this presentation by Dr. Varco. Dr. Robert Mustard (of Toronto) and I have also had some thoughts about reconstruction of the esophagus for slightly different types of lesions. We do not do work with children and have not had this exact problem, but we have had young patients who had lost the use of the esophagus, in whom we wished to establish an alternate route for food. We have been very concerned about by-passing the stomach or about anastomosing the jejunum or any other bowel to the stomach because of peptic ulceration. It seemed to us ideal if we could design a technique to put the new esophagus in the chest and anastomose the lower end to the lower end of the esophagus, if the obstruction were above that level, so as to protect the bowel by normal sphincter action of the lower end of the esophagus. We had one such case in which this procedure was successful. We found that the jejunum had too precarious a blood supply for this maneuver, and we used the colon.

Our patient was a young woman who had a paraffinoma of the mediastinum, secondary to an old extrapleural pneumothorax with paraffin oil fill. She had intractable stenosis of the upper esophagus. We were able to mobilize an isoperistaltic loop of transverse colon pedicled on the middle colic artery, pass it up through the left chest and into the neck, and anastomose the upper end to the upper end of the esophagus, and the lower end to the esophagus just above the diaphragm, so as to by-pass the upper esophagus. She has done well and now has excellent function. I think in cases of this type, if there is a lower segment of esophagus available, which is functional, it is perhaps more ideal from the cosmetic as well as the physiologic point of view to put the new esophagus in the chest and preserve the esophageal sphincter.

DR. MARK RAVITCH, Baltimore.—The problem of peptic ulceration in jejunal loops that have been anastomosed to the stomach is not purely a theoretic one. We have observed this twice, both times in young women who have had an antethoracic esophagoplasty performed for a lye structure. On the other hand we have twice had infants, such as Dr. Varco presented today in his interesting paper, who failed to gain weight and had intractable diarrhea, the stomach having been by-passed, and who did very well and continued to do well after the lower end of the antethoracic jejunal loop was imp'anted into the stomach. It is Yudin's practice to by-pass the stomach routinely. We recently had a visitor from Yugoslavia who, during the Yugoslavian-Soviet honeymoon, spent several years with Yudin, and who has a very large experience of his own in Belgrade—Dr. Papo. His practice is the same, to by-pass the stomach—these are mostly lye and sulfuric acid strictures—anastomosing the loop to the stomach only if diarrhea or malnutrition results, and apparently it can result, even in adults.

One final historical note about Roux's case. That case was reported again forty odd years later by his original first assistant who had taken Roux's last name, possibly having

married Roux's daughter (I have not checked on that) and he is now Picot-Roux. He points out, in a French article, that not only had the patient progressed very well but he had sired two fine children.

One last technical point which is brought to mind by Dr. Kergin's reference to the use of isoperistaltic loops. There are at least two references in the literature to the advantage of reversing the jejunal loop in order to take advantage of the regularly greater size of the first jejunal artery. A careful reading of those papers would indicate that the patients have all succumbed. There is an old reference to Jankouskis of Riga which is carried down in modern literature, and again careful reading of his article shows, in fact, that none of the loops were antiperistaltic, they were all isoperistaltic.

SURGICAL TREATMENT FOR CONGENITAL AORTICOPULMONARY FISTULA

Experimental and Clinical Aspects

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Baltimore, Md.

A DEVELOPMENTAL defect in the aortic septum with communication between the aorta and the pulmonary artery near the base of the heart is a rare congenital anomaly. Gasul, Fell, and Casas,[1] who reported a case of this type in 1951, were able to find only twenty-four other cases recorded in medical reports during the last century. Little is known about the origin of the anomaly except that it probably results from an arrest in the development of the aortic septum between the fifth and eighth weeks of intrauterine life.[2] In the normal fetus the growth of the aortic septum divides the primitive truncus arteriosus into two separate vessels, the aorta and the pulmonary artery. Failure of growth or of fusion of the anlagen of the septum may result in persistence of certain features of the truncus arteriosus in postnatal life. Depending on the degree of the arrest, the windowlike communication between the aorta and the pulmonary artery which results may vary from a small round opening 5 mm. or so in size located 8 or 10 mm. above the cusps of the pulmonary valve to a large defect which extends from the valve cusps to the bifurcation of the main pulmonary artery (Fig. 1).

Congenital aorticopulmonary fistula is probably the best descriptive term for this anomaly from both the physiologic and clinical points of view. The alterations in circulatory dynamics associated with it appear to be similar to those produced by persistent patency of the ductus arteriosus. The clinical syndrome has been discussed recently by Spencer,[3] Gibson,[4] Gasul,[1] Dammon,[5] and their respective associates.

Our interest has been primarily concerned with the development of a method of surgical treatment for congenital aorticopulmonary fistula. The purpose of this article is to describe experimental techniques for production and closure of aorticopulmonary fistula and to report the successful clinical application of the method of closure in an infant.

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EXPERIMENTAL ASPECTS

Production of Aorticopulmonary Fistulas.—The object of these experiments was to produce a laboratory preparation which would resemble anatomically and physiologically congenital aorticopulmonary fistula in patients. Several years ago during the development of the technique of creation of an artificial ductus arteriosus, Dr. Alfred Blalock devised a means of producing an aorticopulmonary fistula which consisted of placing a stab wound between the pulmonary artery and the aorta near the base of the heart where the walls of the two vessels are in apposition. This technique did not prove to be entirely satisfactory, however, and it was found by Blalock and his co-workers that fistulas produced in this way tended to close spontaneously.[6]

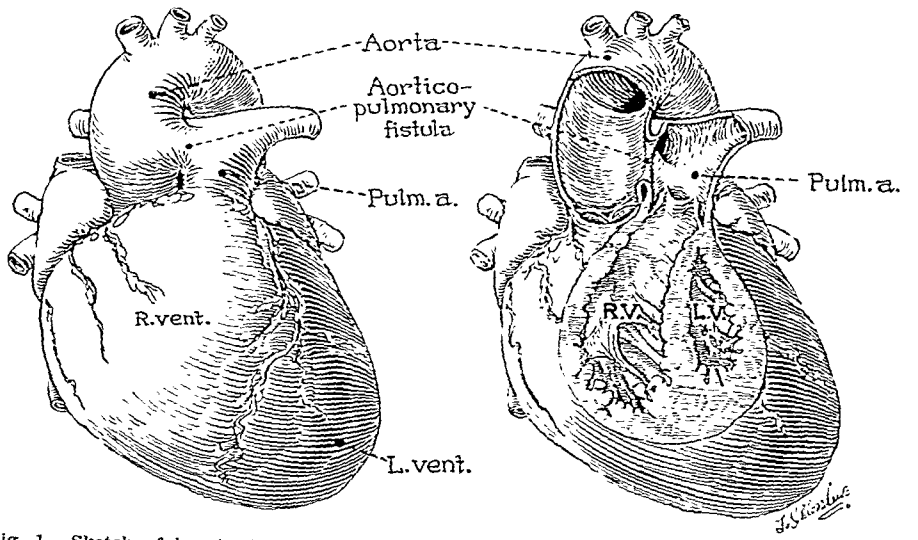


Fig. 1.—Sketch of heart at autopsy on 18-month-old baby with congenital aorticopulmonary fistula exhibiting typical anatomic features of the anomaly.

The method adopted for use in the present experiments is the construction of a suture anastomosis between the ascending aorta and the main pulmonary artery. It is essentially a modification of the technique of lateral anastomosis between the descending aorta and the left pulmonary artery described by Potts, Smith, and Gibson.[7]

Twenty-five adult mongrel dogs were used in these experiments. The procedure which was employed in the production of aorticopulmonary fistula in these animals was as follows. After morphine and atropine premedication, the animal was anesthetized with ether. Pulmonary ventilation was maintained by the use of an intratracheal tube and a mechanical insufflator.[8] Using aseptic technique an incision was made in the fourth left intercostal space. The pericardium was opened widely. The intrapericardial fat pad overlying the base of the aorta and the pulmonary artery was excised and both vessels were carefully freed of investing areolar tissue down to their points of origin

from the heart. With the vessels widely mobilized, a curved Potts ductus clamp was applied to the anteroinferior aspect of the ascending aorta so as to isolate a crescentic segment of the aortic wall. A similar segment of the wall of the adjacent main pulmonary artery was isolated by the application of a straight Potts ductus clamp to its anterosuperior aspect. Each clamp was applied in parallel to the long axis of the vessel. A small incision was then placed in the wall of each isolated vascular segment and a lateral anastomosis constructed with 00000 arterial silk sutures by the Blalock method.[9] After the removal of the clamps a thrill was palpable in the pulmonary artery. The pericardium was closed loosely, the lung re-expanded, and the chest wall closed in layers. The steps in technique are illustrated in Fig. 2.

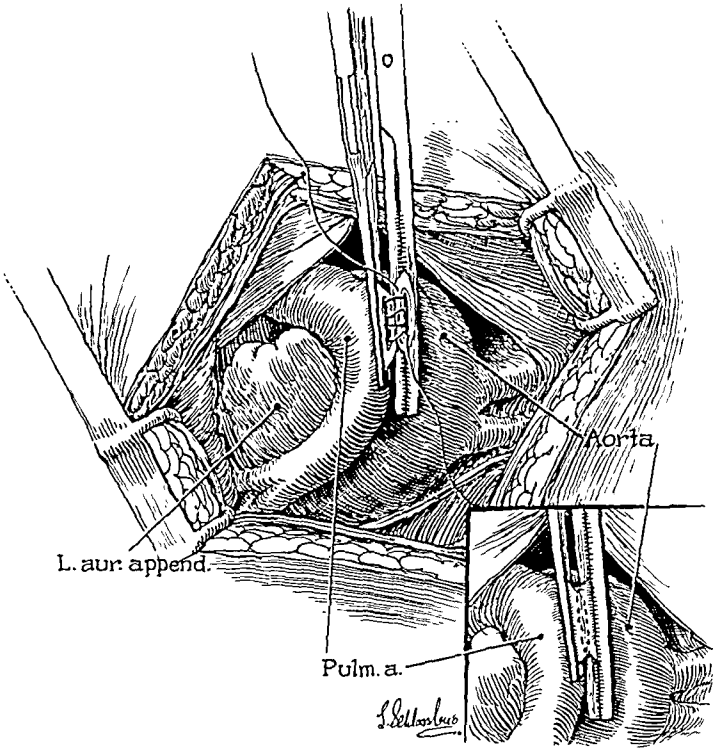


Fig. 2.—Technique of production of experimental aorticopulmonary fistula.

With this method it is possible to place the anastomosis within one centimeter or less of the aortic valves and even closer to the pulmonary valves. A typical example of a small fistula produced by this technique is seen in Fig. 3.

Three of the twenty-five dogs used in these experiments died of hemorrhage from the anastomosis in the immediate postoperative period. In each of the remaining dogs an aorticopulmonary fistula was successfully produced by the method described. A continuous murmur with systolic accentuation could be heard in the postoperative period in each case. The size of the fistula

which was produced in these animals varied from 4 to 10 mm. in diameter. The smaller fistulas (4 to 5 mm. in diameter) were well tolerated by the animals and there were no manifestations of cardiac failure during observation periods of one to four months in this group. Animals with aorticopulmonary fistulas measuring 6 to 10 mm. in diameter died with pulmonary edema in four to ten days after operation.



Fig. 3.—Photographs of dog's heart showing aortic and pulmonary aspects of small experimental fistula one month after its production.

Closure of Aorticopulmonary Fistulas.—In twelve of the dogs with experimental aorticopulmonary fistulas a second thoracotomy was done and an attempt was made to close the fistula. Because of the windowlike nature of aorticopulmonary fistulas in patients and the relatively large diameter of some of these congenital communications, it was felt that a technique of closure based on the use of encircling ligatures or suture ligatures would prove to be unsatisfactory in clinical application. Consequently, in these animals with experimental fistulas efforts were directed toward closure of the fistula by a method of division and suture. The procedure used was as follows. The animal was anesthetized with ether and the old thoracotomy wound was reopened. Adhesions between the lung and the pericardium were separated and the pericardium was opened, exposing the great vessels and the site of the fistula. Careful dissection was carried out to free the circumference of the aorta and

the main pulmonary artery, proximal and distal to the fistula. With the vessels mobilized as widely as possible the dissection was then carried around the fistula itself. A curved Potts ductus clamp was applied to the aortic side of the fistula grasping a crescentic segment of the aortic wall. In a similar manner a straight Potts ductus clamp was applied to the pulmonary artery so as to occlude the pulmonary aspect of the fistula. The clamps were applied in parallel to the long axis of the vessels. The fistula was divided between the clamps so as to leave a vascular cuff for suture within the grasp of each clamp. Closure of the defect in the wall of each vessel was accomplished with a running mattress suture of 00000 arterial silk which was returned on itself as a continuous over-and-over suture. The clamps were then removed, the pericardium was closed loosely, the lung re-expanded, and the chest closed. Fig. 4 illustrates the steps in closure by this method.

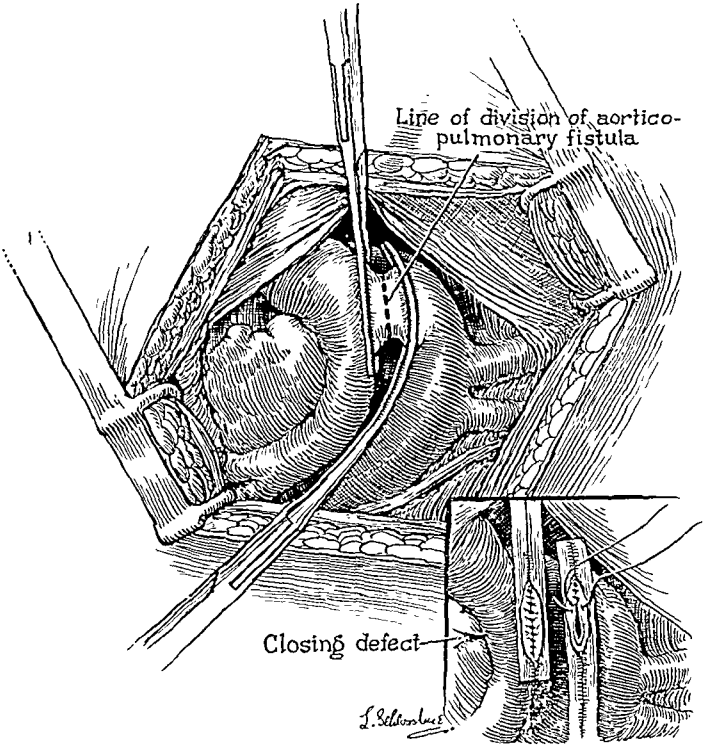


Fig. 4.—Technique of closure of experimental aorticopulmonary fistula by division and suture.

There are several possible variations in the method of application of the clamps and in the choice of straight and curved Potts forceps. However, the use of the curved forceps on the aortic side of the fistula permits a larger segment of aortic wall to be more easily grasped than when a straight clamp is used for this purpose.

Successful closure of the experimental aorticopulmonary fistula by this method was accomplished in five of the twelve dogs in which it was attempted.

The other animals were lost from hemorrhage at the time of attempted closure. Dense scar makes the dissection extremely difficult several months after the fistula is produced, and if closure is attempted within the first week or two after the anastomosis, friable granulations and easy dehiscence of the anastomosis add to the technical hazard. With a single exception, the successful closures were all accomplished one to three months after production of the fistula. These animals have been observed from one to six months since the closure of their experimental aorticopulmonary fistulas and have remained healthy and active. There have been no apparent ill effects from the slight degree of narrowing of the aorta and the pulmonary artery which has undoubtedly resulted from the production and closure of the fistula in the survivors.

CLINICAL ASPECTS

During an eighteen-month period (1950 to 1951) three patients with congenital aorticopulmonary fistula were studied and subsequently submitted to thoracotomy at the Johns Hopkins Hospital. The diagnostic problem presented by these patients and a detailed description of the clinical syndrome of aorticopulmonary fistula exceed the scope of this article and will be reported separately by Taussig and her associates.[10]

Cardinal clinical findings in the first two patients of this small series included absence of cyanosis and polycythemia, progressive cardiac enlargement, and a loud systolic murmur over the precordium, maximal in the third or fourth intercostal space to the left of the sternum. Cardiac catheterization indicated the presence of a large left to right shunt. A diagnosis of "atypical" ductus arteriosus was made in each case, although high interventricular septal defect and congenital aorticopulmonary fistula could not be excluded.

At operation in the first patient (female, aged 18 months) an aorticopulmonary fistula was found which measured 10 mm. in diameter (Fig. 1). The lower margin of the fistula lay 6 or 7 mm. above the pulmonary valves. During the dissection around the fistula, the base of the pulmonary artery was lacerated and the ensuing severe hemorrhage could not be controlled. At autopsy the fistula proved to be an isolated cardiac anomaly.

In the second patient (male, aged 4 years) thoracotomy revealed the malformation illustrated in Fig. 5. There was a right aortic arch and a large defect in the aortic septum which seemed to extend down to the level of the arterial valves. There was no sulcus at the base of the large common arterial trunk to indicate a point of demarcation between pulmonary artery and aorta. Direct measurements of pressure in the aorta and the pulmonary artery showed identical readings. No attempt was made to close this huge fistula.

Experience with these two patients stimulated our interest in devising a method of surgical treatment for congenital aorticopulmonary fistula and prompted the development of the experimental techniques which have been described. In the third patient the technique of closure of aorticopulmonary fistula by division and suture was applied successfully.

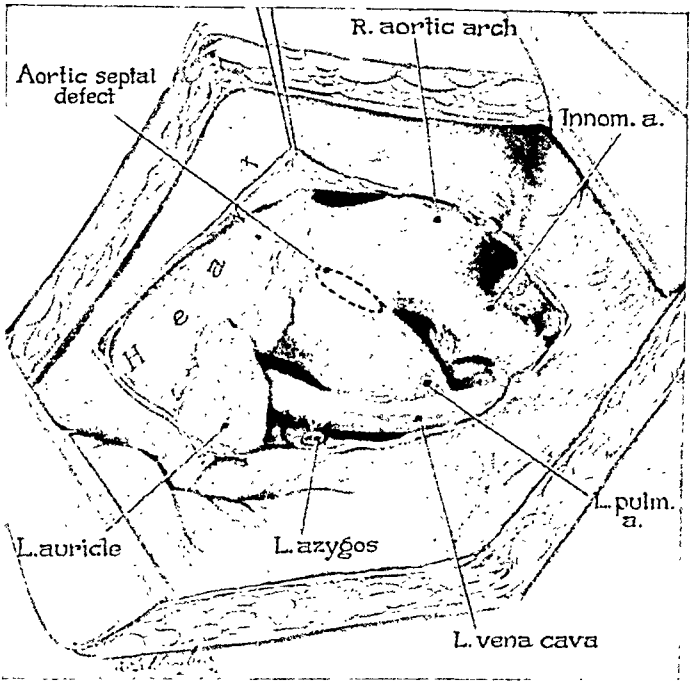


Fig. 5.—Sketch of findings at operation in 4-year-old boy with right aortic arch and large aorticopulmonary fistula which apparently extends to the level of the valves. Closure was not attempted.



Fig. 6.



Fig. 7.

Fig. 6.—Reproduction of posteroanterior roentgenogram of chest before operation in 10-month-old infant with congenital aorticopulmonary fistula (patient D. S.)

Fig. 7.—Reproduction of preoperative retrograde thoracic aortogram in patient D. S., demonstrating aorticopulmonary fistula (left anterior oblique view).

CASE REPORT

D. S., a 10-month-old white female, was admitted to the Harriet Lane Home of The Johns Hopkins Hospital on Dec. 5, 1951, because of cardiac failure. The mother's pregnancy had been uneventful. Delivery occurred two weeks prior to term and the birth weight was 5 pounds, 9 ounces. Since the age of 2 months the infant had been followed in the clinic because of recurrent respiratory infection, dyspnea, tachycardia, and progressive cardiac enlargement. She had always been a feeding problem and failed to make a satisfactory gain in weight. Cyanosis had never been noted.

Physical Examination.—On admission examination revealed a thin, underdeveloped female infant with temperature 97°, pulse 130, respiration 40, and blood pressure 130/60. Head, eyes, ears, nose, and throat were normal. There were fine moist râles at both lung bases. The heart was enlarged to the right and left. The apical impulse was in the fifth left intercostal space in the anterior axillary line. There was a diffuse, grade 2 systolic murmur along the left sternal border, maximal in the fourth left intercostal space. The liver edge was palpable 3 cm. below the costal margin. There was no cyanosis, clubbing of the digits, or edema.

Fluoroscopy.—The heart was moderately enlarged (Fig. 6). The pulmonary conus was not prominent in appearance, but was rather more active than normal. The pulmonary hilar shadows were moderately increased and showed slight but definite pulsations. The peripheral lung fields were not remarkable. There was a left aortic arch.

Angiocardiogram.—Four cubic centimeters of 70 per cent Diodrast was injected into a vein in the right arm. Both anteroposterior and lateral exposures were made and films were taken at 1, 2, 3, 4, 5, 6, and 7 seconds. The contrast medium was seen to enter the right auricle through a normal superior vena cava. The right auricle was not enlarged. The right ventricle seemed to be of normal size and the pulmonary arteries received a normal amount of dye. The aorta was not visualized. No patent ductus was demonstrated.

Retrograde Thoracic Aortogram.—The left ulnar artery was cannulated and films were taken following the retrograde injection of 5 c.c. of 35 per cent Diodrast. On one film the entire thoracic aorta was well visualized (Fig. 7). The aorta was of normal size and contour. The contrast medium had regurgitated well down into the ascending aorta and there was definite opacification of the main trunk of the pulmonary artery and its branches, indicating the presence of an abnormal communication between the aorta and the pulmonary artery. No patent ductus could be identified.

Cardiac Catheterization.—Catheterization was attempted but because of technical difficulties the superior vena cava could not be cannulated. The inferior vena cava was entered via the saphenous vein but the development of a nodal rhythm necessitated the termination of the procedure.

Laboratory Studies.—Red blood count 4.71 million, hemoglobin 12 Gm. per cent, hematocrit 37.2. Electrocardiogram showed a rate of 100 with a sinus rhythm, P-R interval 0.18 seconds, QRS interval 0.02 second. The electrocardiogram was interpreted as showing a balanced axis with no definite ventricular hypertrophy and slight digitalis effect.

Course.—Despite digitalization the infant remained in cardiac failure with progressive enlargement of the heart and of the liver. After a review of all the data, Dr. Taussig and her associates made a diagnosis of congenital aorticopulmonary fistula and operation was advised.

Operation.—Operation was carried out by one of us (H. W. S., Jr.), Dec. 21, 1951.

Anesthesia.—Intratracheal oxygen-ether was used.*

Technique.—A cannula was inserted in an ankle vein for constant venoclysis and the chest was prepared and draped for bilateral anterior thoracotomy. A left submammary incision was placed, and the chest entered in the third intercostal space. Mediastinal dissection revealed no evidence of a patent ductus. There was a systolic thrill in the

*Administered by Miss Olive Berger.

pulmonary artery. The pericardium was opened widely and it was immediately apparent that there was a large fistula between the aorta and the pulmonary artery near the base of the heart (Fig. 8).

In order to obtain a more adequate exposure the cutaneous incision was extended across the sternum and over the third intercostal space on the right to a point just medial to the nipple. The sternum was divided with a Gigli saw, the internal mammary vessels were ligated and divided, and the entire wound was opened well into the right pleural cavity. A rectangular flap of pericardium was reflected to afford a wide exposure of the

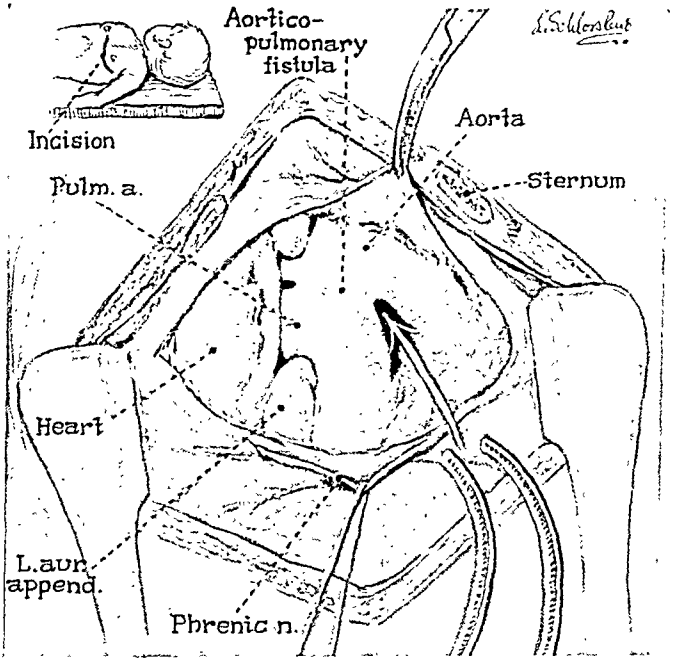


Fig. 8.—Sketch of anomaly as exposed at thoracotomy in patient D. S.

great vessels at the base of the heart. The aorta was normal in size but the pulmonary artery was larger than normal. There was a fistula between the aorta and the pulmonary artery which measured 16 mm. in its external diameter. The lowermost extent of the fistula lay 5 mm. above the base of the pulmonary artery and approximately 12 to 15 mm. from the base of the aorta. With the operator's finger in the transverse sinus, compression of the fistula bimanually readily delineated the elliptical margins of the communication between the two vessels and obliterated the thrill in the pulmonary artery. A careful dissection was then carried out to free the adventitia and pericardial reflexion from the aorta and pulmonary artery proximal and distal to the fistula. During this phase of the procedure there were two transitory episodes of asystole which responded promptly to the intraventricular injection of 2 c.c. of 10 per cent calcium chloride solution and cardiac massage. After the great vessels had been mobilized as widely as possible the dissection was carried in the plane anterior to the origin of the right pulmonary artery and posterior to the fistula so as completely to encircle the latter. A curved Potts ductus clamp was then passed into position on the aortic side of the fistula in such a way as to grasp a generous segment of the wall of the ascending aorta. A second curved Potts ductus clamp was placed in parallel with the first clamp and applied to the main pulmonary artery so as to occlude the pulmonary aspect of the fistula. Both clamps were closed to the third

ratchet and locks were applied. The fistula was divided between the clamps, and the defect in the aortic wall was closed with a continuous over-and-over suture, using 00000 silk on an atraumatic needle (Deknatel) in two rows. Upon removing the aortic clamp there was no bleeding. The very short cuff in the grasp of the clamp on the pulmonary artery was improved by applying a second curved clamp and removing the original one. The defect in the wall of the pulmonary artery was then closed with a continuous mattress suture of 00000 silk which was returned on itself as an over-and-over suture. There was no bleeding on removing the clamp (Fig. 9).

After the completion of these maneuvers the infant's heart action seemed to be improved and no thrill could be palpated in the aorta or the pulmonary artery. The pericardium was closed loosely. Catheters were placed in both pleural cavities and led out through stab wounds for water-seal drainage. The divided sternum was re-approximated with mattress sutures of chromic catgut, the lungs were re-expanded, and closure of the thoracotomy wound was carried out in layers.

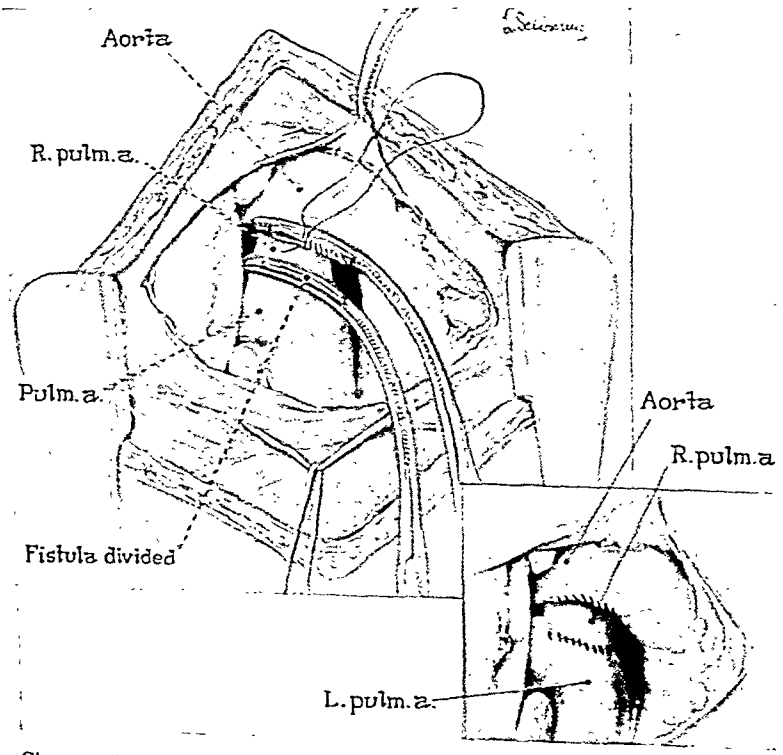


Fig. 9.—Closure of congenital aorticopulmonary fistula by disvision and suture using curved Potts ductus forceps (patient D. S.).

Postoperative Course.—The early postoperative course was characterized by moderate fever and tachycardia. The infant was maintained on penicillin, terramycin, and digitalis. After the first week the fever subsided, the infant became very active, took feedings well, and began to gain weight. With the disappearance of manifestations of cardiac failure digitalis was discontinued. The final roentgenogram of the chest prior to discharge showed some reduction in the size of the heart and clear lung fields. The patient was discharged from the hospital on Jan. 5, 1952. Since discharge she has been followed in the Cardiac Clinic by Dr. Taussig and her associates. In the five months since leaving the hospital the

baby has continued to do well with no manifestations of decompensation or other cardiac difficulties. A soft systolic murmur persists which is thought to be functional.

COMMENT

The experiments which have been described demonstrate the feasibility of constructing a suture anastomosis between the ascending aorta and the main pulmonary artery. The technique of producing an aorticopulmonary fistula might be suggested as an alternative to the Blalock and Potts anastomoses as a means of increasing the blood flow to the lungs in patients with tetralogy of Fallot. However, used in this capacity it would have no particular advantage over the other methods and would be a considerably more hazardous undertaking. The procedure has served to establish a satisfactory laboratory preparation which resembles congenital aorticopulmonary fistula in patients. In these experiments the animals exhibited a high degree of tolerance during the fifteen- to thirty-minute period of the anastomosis to degrees of partial occlusion of the ascending aorta and the main pulmonary artery ranging from 30 to 50 per cent. None of the animals showed a tendency to spontaneous closure of the fistula during the one- to four-month period of observation permitted by these experiments. Studies will be made to assess the long-range effects of this type of fistula and to investigate the relation between the size of the fistula and the volume of the left to right shunt.

The method of closure by division between Potts ductus clamps is simply an adaptation of Gross's original technique for division of patent ductus arteriosus[11] to the closure of an arterial fistula of the "window" type. In both the production and closure of the experimental fistulas and in the successful clinical application of the method of closure the Potts ductus clamps[12] have proved to be invaluable. The multiple fine teeth of these ingenious instruments will hold securely a segment of arterial wall without damage to the vessel. A narrow-bladed clamp of the Potts type, which offers a great advantage in the division of a short ductus arteriosus, is a necessity in the closure of an aorticopulmonary fistula by division and suture.

In the majority of the reported cases of this malformation in which an adequate description of the anomaly is included, the fistula has been quite similar anatomically to that which we have been able to close. The average diameter of the communication in most instances was in the range of 10 to 12 mm. The inferior margin of the fistula was usually several millimeters above the level of the pulmonary valve. Thus, it seems that the majority of congenital aorticopulmonary fistulas are amenable to surgical closure by the method which has been described. This method does not seem to be applicable without extreme risk to the closure of a huge fistula of the type which extends down to the level of the arterial valves as described by Dadds and Hoyle[13] or to the anomaly illustrated in Fig. 4. The presence of a sulcus, however small, which demarcates the lower extent of the fistula and permits dissection to be carried completely around the latter, is of considerable technical significance.

Surgical closure of congenital aorticopulmonary fistula in individual cases has been accomplished recently by Gross[14] and by Varco[15] by methods of liga-

tion or suture-ligation. The technique of division and suture would appear to offer a more accurate means of correction of a large windowlike communication between the great arterial trunks with less tendency to recurrence of the fistula. While undoubtedly rare as compared to patent ductus arteriosus, congenital aorticopulmonary fistula probably has a much greater incidence than indicated by the few reported cases. It is likely that the malformation will be recognized more frequently in the future with the current trend toward careful diagnostic evaluation of all forms of congenital heart disease.

SUMMARY

Congenital aorticopulmonary fistula is a rare cardiac malformation which results from an arrest in the development of the aortic septum. In an effort to devise a method of surgical treatment, the anomaly was reproduced in dogs. A technique for closure of the experimental fistulas by division and suture was worked out in the animals. This technique has been applied successfully in the closure of a 16 mm. aorticopulmonary fistula in a 10-month-old infant. Experimental and clinical aspects are briefly discussed.

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DISCUSSION

DR. AARON HIMMELSTEIN, New York.—Our group has been interested in the diagnosis of this lesion, particularly in differentiating it from the atypical patent ductus in which the physical findings are unusual. I would like to make the comment that in the catheterization studies, if one is able to thread a catheter through an aorta-pulmonary artery septal defect, it will go up the arch of the aorta and take the route that the arch

of the aorta takes, whereas if one threads a catheter successfully through a patent ductus it does not go through the arch of the aorta, but subtends that arch and goes down the descending aorta. This may be a feature that would help in the differential diagnosis of these two conditions.

DR. ROSS ROBERTSON, Vancouver.—I am intensely interested in Dr. Scott's excellent paper, because in operating on forty-five patients suspected of having patent ductus arteriosus I have encountered four similar cases. Regarding the name, I would like to take issue with Dr. Scott, possibly because I am a Canadian and favor Maud Abbott's terminology of aortic septal defect, which is a defect in the septum which divides the truncus arteriosus into the aorta and the main pulmonary artery.

I do not feel quite as sanguine about the preoperative diagnosis as Dr. Scott. I admire him for making the diagnosis in this case. Even postoperatively we have been unable to distinguish the cases definitely, in a clinical way, from a patent ductus, the reason being, of course, that they have the same physiologic effect as a patent ductus. We have come to feel that if you have a case of patent ductus which seems to be almost too good to be true, then you suspect this defect. If you have a child with symptoms of dyspnea, or undernourishment, or excess fatigue, and find an enlarged heart with a very loud continuous murmur placed a little more medially than that of a patent ductus; if you have a very high pulse pressure with the diastolic pressure frequently falling to zero; and on x-ray examination, in addition to the large heart you see that the pulmonary vascular markings are greatly accentuated—then, I think, you should suspect this defect.

When we encountered this lesion at operation, there was no patent ductus, but there was a thrill over the main pulmonary artery close to the heart. On opening the pericardium the thrill was found to originate about one inch from the heart; and the main pulmonary artery and aorta seemed to be fused together. Subsequently, suspecting this defect, we produced a similar defect on a dog and did aortograms which demonstrated the defect. Aortograms on one of these children showed the same type of defect as produced in the dog. We have not had the courage to proceed any further, suspecting that the defect continued down to the pulmonary valves. I am grateful to hear Dr. Scott describe the little notch found between the defect and the pulmonary valve and permitting the application of forceps for repair.

DR. ROBERT SHAW, Dallas.—About three years ago we saw a 5-year-old boy from a neighboring community who obviously had a congenital heart lesion. On fluoroscopy we learned that this child had enormous pulmonary arteries with a typical hilar dance. Angiocardiography was not very conclusive but our radiologist felt that the diagnosis was probably a large interatrial septal defect, although there were some features in the case that did not seem to be explained by this diagnosis. We advised the parents that as yet we had no surgical procedure we could offer the child. About a year and a half later this child suddenly died while playing in the back yard of his home, and at autopsy it was found that he had an aorticopulmonary fistula, and that there had been rupture of the pulmonary artery intrapericardially with sudden tamponade.

DR. FRANK GERBODE, San Francisco.—I wish to compliment Dr. Scott upon his approach to this very difficult problem. The experimental method he used in solving it is, I think, the one we should adopt more often in the solution of cardiovascular problems. We have encountered fistulous connections near the base of the aorta in five patients in whom exploration was carried out for patent ductus arteriosus. In each instance there was no ductus. One of these patients subsequently expired, and at necropsy we found a ruptured aneurysm of the sinus of Valsalva. (Slide.) This slide shows a small connection just above the valve where there was a fistula leading to the right ventricle. This produced a continuous murmur and blood pressure changes, like a ductus. Just beneath this there is a high septal defect which also contributed to the continuous murmur.

In our cases cardiac catheterization was not of great help, although it did show a higher oxygen saturation of the pulmonary arterial blood than is usual with a ductus. In

other words, the differences were greater than one might expect with a normal ductus. In general, the continuous murmur was louder than it is with a ductus. We were unable to close the opening in any of these patients because of the proximity of the connection to the coronary vessels or to the valves. I think this must be borne in mind in all these patients, because if one tries to close an opening low in the great vessels, one will encounter this difficulty. In Dr. Scott's case it was possible to establish the proximal limit of the opening. This case will serve to stimulate further efforts toward developing methods of closing these defects.

DR. SCOTT (Closing).—I would like to thank the discussants for their interesting remarks. Dr. Himmelstein's use of the catheter certainly should prove to be an excellent means of diagnosis if one is lucky enough to enter the fistula. I am very much impressed by the large incidence of this hitherto considered rare anomaly exhibited by the remarks of the discussants. I think there is one thing about the technical side of the attempts at closure of these defects that might be brought out; that is, that in the two cases we had an opportunity to examine, namely, the one at autopsy and the clinical case which was successfully divided, there was a difference in the proximity of the fistula to the aortic annulus and the annulus of the pulmonary valve. In other words, the base of the aorta lies lower than does the base of the pulmonary artery, so that the coronary arteries at their points of origin will be somewhat below the fistula if the fistula is associated with some kind of little sulcus to demarcate its lower extent. I believe this is an important technical point.

PULMONARY FUNCTION AFTER MULTIPLE SEGMENTAL RESECTION FOR BRONCHIECTASIS

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BOSTON, MASS.

BRONCHIECTASIS may involve any of the eighteen segments which comprise the five lobes of the two lungs. The disease is frequently bilateral, but rarely is there involvement of all the constituent segments of any upper or lower lobe. The right upper lobe, the two top segments of the left upper lobe, and the superior segments of both lower lobes are seldom infected and usually compensate in size and function.

Severity of symptoms is not necessarily related to the extent of involvement, but relief of these symptoms is dependent upon the complete excision of the disease. The conservation of normal healthy tissue is as much a part of the fundamental concept of surgery as the eradication of the disease. The intent of this basic concept is achieved by segmental resection in surgery for bronchiectasis. Segmentectomy presents the problem of greater technical difficulty and more diligent postoperative attention, but the ultimate advantage of preserving healthy pulmonary tissue makes it desirable for the unilateral case as well as the severe bilateral case.

The purpose of this paper is to present the results of postoperative pulmonary function studies on patients with segmental resection for bilateral and unilateral bronchiectasis.

METHOD OF STUDY

The results of this investigation comprise data from a group of fourteen unselected patients who had either unilateral or bilateral segmental resection one to seven years prior to study. None of these patients had preoperative pulmonary function studies. All of them were admitted to the hospital for study. Pulmonary physiologic determinations were performed in the fasting state. Total spirometry was done in the morning and differential bronchospirometry in the afternoon. Arterial bloods were drawn for gas analyses and pH determinations during the resting state. On another day, total lung volume measurements were obtained.

Read at the Thirty-second Annual Meeting of The American Association for Thoracic Surgery at Dallas, Texas, May 8-10, 1952.

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Ventilation Studies.—Total vital capacity was performed with modified Benedict-Roth spirometer, and the timed capacity was measured with a vital capacity spirometer.[1] Oxygen consumption, tidal volume, respiratory minute volume (R.M.V.), inspiratory capacity, and expiratory reserves were obtained using a balanced spirometer with soda lime in the circuit. The maximum breathing capacity (M.B.C.) was obtained using a Hans-Rudolph high velocity respiratory valve and a Douglas bag.[2] The gas was collected for thirty seconds, measured by a Tissot spirometer, and reported in liters per minute at 37° C. wet. The best of three efforts is reported. The walking ventilation test consisted of a level walk at the rate of 180 feet per minute for three minutes.[3] The expired air was collected in a Douglas bag; the volume was measured like that of the M.B.C. and recorded in liters per minute at 37° C. wet. The walking index was calculated from the equation: $\frac{\text{walking ventilation}}{\text{M.B.C.}} \times 100$.[3] The predicted M.B.C. and vital capacity (V.C.) were calculated according to Cournand's regression formulas.[4] The air velocity index was calculated from the equation: $\frac{\text{per cent of predicted M.B.C.}^{5}}{\text{per cent of predicted V.C.}}$. The ventilation equivalent for oxygen was calculated from the equation: $\frac{\text{minute ventilation in liters}}{\text{minute oxygen uptake in c.c.}}$ This is reported as the number of liters ventilated per 100 c.c. oxygen removed. The breathing reserve may be obtained from the equation: $\frac{(\text{M.B.C.}-\text{M.V.})\ 100.}{\text{M.B.C.}}$[6]

Differential Bronchospirometry.—The patients were sedated with 75 mg. pentobarbital sodium and 0.6 mg. scopolamine thirty minutes before the start of the procedure. Laryngo-tracheo-bronchial anesthesia was obtained with 4 per cent cocaine. A Carlens double-lumen catheter was inserted under fluoroscopic guidance.[10] Records of oxygen consumption, tidal exchange, respiratory minute volume, inspiratory reserve, expiratory capacity, and vital capacity were obtained on a twin recording spirometer.

Arterial Blood Studies.—Arterial oxygen content, capacity, and carbon dioxide determinations were made on 1 c.c. samples of whole blood of the method of Van Slyke and Neill.[7] The arterial blood for pH was determined by a Beckmann model G glass electrode. The pH was corrected for a temperature of 38° C.[8] The carbon dioxide tension was calculated from the carbon dioxide content in volume per cent and the pH value from the nomogram of Peters and Van Slyke.[9] Total carbon dioxide values in millimeters per liter were read from the chart of Peters and Van Slyke.[10]

Total Lung Volume.—The patient was allowed to rest in the supine position for thirty minutes previous to the measurement of function residual capacity (F.R.C.). The Darling[11] open circuit with slight modification was used. After the first determination, the inspiratory capacity, residual capacity, and vital capacity were recorded on a Warren Collins respirometer. After a two-hour interval of rest, these tests were repeated. The following calculations were

made from these determinations: functional residual capacity, residual volume, total capacity, and alveolar nitrogen content after seven minutes of oxygen breathing.

CASE HISTORIES

These four cases serve to illustrate the value of segmentectomy in bronchiectasis by eradication of disease and conservation of healthy pulmonary tissue.

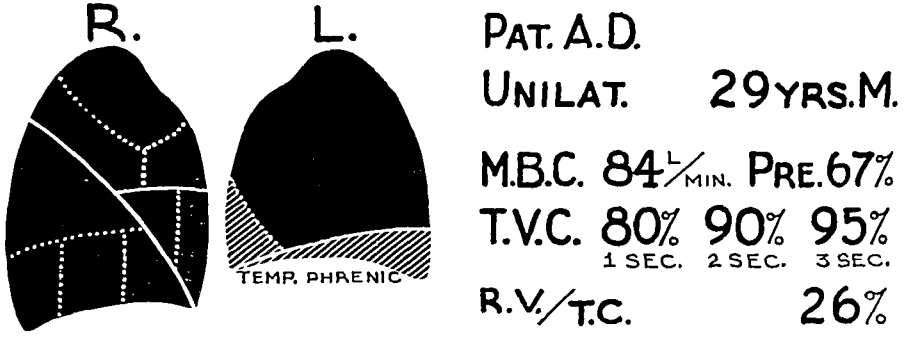
CASE 1.—Patient A. D. had unilateral bronchiectasis with a chronic abscess since the age of 3 years, which involved the left lower lobe, the lingula, and anterior segments of the left upper lobe. The involved segments were excised in January, 1952.

The studies in this patient are of fundamental significance because they provide the opportunity to evaluate the function of an individual bronchopulmonary segment.

The maximum respiratory muscular effort (M.B.C.) of 84.4 L. per minute, 67 per cent predicted, indicates that the total function is efficient for the total number of remaining segments.

This patient had a total lung capacity of 3.8 L., and the residual volume was 26 per cent of his total capacity. This indicates that emphysema is not present. The alveolar nitrogen index of intrapulmonary mixing is normal, further evidence of normally functioning lung tissue without emphysema.

The functional relationship between the one segment on the left and the ten segments of the right lung show that the one segment contributes 22.6 per cent of the total oxygen consumption and 28 per cent of the ventilation. This clearly demonstrates the value of conserving one segment which could be lifesaving in any respiratory emergency.



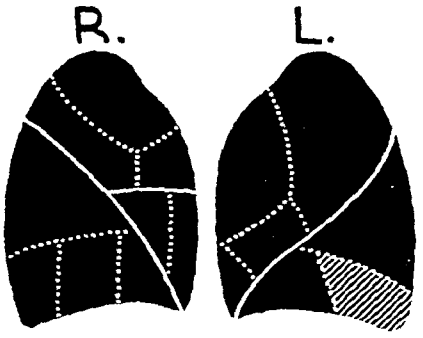
	RIGHT 10 SEG.	LEFT 1 SEG.
O_2 Cons. %	77.4	22.6
Vent. %	72	28
V.C. %	80	20
Vent. Eq. L./min.	2.4	3.2

Fig. 1.

CASE 2.—Patient N. F. had bronchiectasis involving the posterior and lateral basal segments of the left lower lobe. An empyema at the left base had been drained in 1930. Following this, he developed a cough and occasionally some purulent sputum, and on three occasions he had minimal drainage through the previous operative site. The involved segments were removed, and postoperatively there was a moderate air leak from the segmental surfaces for forty-eight hours.

The pertinent problem in this case was either to resect the entire left lower lobe or conserve the normal segments and remove only the diseased tissue. The removal of the two abnormal segments allowed the remaining segments of that lobe to participate with the left

upper lobe in 36.6 per cent of the total oxygen consumption. The left hemidiaphragm was elevated and fixed due to surgery for empyema in 1930, and this may explain the decrease in vital capacity and the elevated ventilation equivalent as compared to the right lung. Residual volume and intrapulmonary mixing volume were normal. The removal of two additional normal segments most likely would have further impaired the function of the left lung.

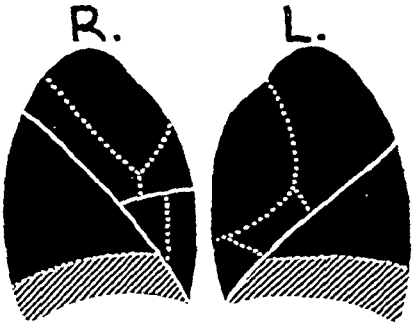


	RIGHT 10 SEG.	LEFT 6 SEG.
O_2 Cons. %	63.4	36.6
Vent. %	59.1	40.9
V.C. %	68.8	31.2
Vent. Eq. L./min.	4.64	5.56

Fig. 2.

CASE 3.—Patient G. Z. was markedly incapacitated from severe bilateral basal bronchiectasis since early childhood. The left basal segments were excised in February, 1950, and the right basal segments in June, 1950. There were no postoperative complications.

The resection of only the diseased segments has not impaired pulmonary function. This is well *demonstrated* by total function studies, differential bronchospirometry, and arterial blood gases.

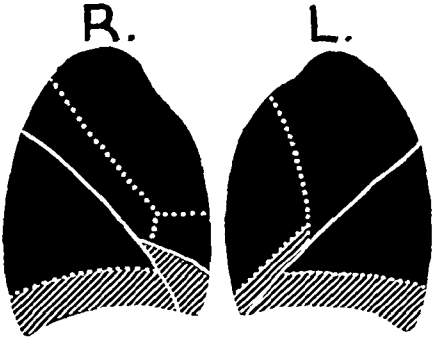


	RIGHT 6 SEG.	LEFT 5 SEG.
O_2 Cons. %	47	53
Vent. %	54	46
V.C. %	55	45
Vent. Eq. L./min.	5.47	4.1

Fig. 3.

CASE 4.—Patient H. K. had the typical bilateral bronchiectatic pattern—basal segments bilaterally, right middle lobe, and lingula. In addition to severe cough, excessive amounts of purulent sputum, and dyspnea, she had severe recurrent pulmonary hemorrhages. These symptoms developed at the age of 14 or 15 years. The abnormal segments on the right were removed in October, 1948, and those on the left in April, 1949. The postoperative course was normal.

The M.B.C. of 54 L. per minute on this patient with only seven pulmonary segments may be considered as evidence of good ventilatory function. The timed vital capacity reveals normal speed of air movement. Although the residual volume is 29 per cent of total capacity, the intrapulmonary mixing index is within normal range. This indicates that the patient has some overdistention or compensatory emphysema without impairment of gas distribution. The differential bronchospirometry indicates essential normal distribution of oxygen consumption, ventilation, and V.C. among the remaining segments. The left lung, however, ventilates more efficiently than the right.



	RIGHT 4 SEG.	LEFT 3 SEG.
O_2 Cons. %	58.2	41.8
Vent. %	67.3	32.7
V.C. %	60	40
Vent. Eq. L./min.	3.1	2.1

Fig. 4.

RESULTS

The total results of this study are summarized in Tables I, II, and III. In the group with unilateral resection, the total function studies show that the fewer the number of segments excised the greater the percentage predicted of M.B.C. The walking ventilation, W.V./M.B.C. ratio, ventilation equivalent, and the speed of air flow on expiration in all the cases of this series were within normal limits.

A comparison of the function of the normal lung and that of the operative side (Table III) illustrates the following:

1. The percentage of oxygen consumption and ventilation increases with the number of segments conserved.

2. Ventilation was slightly increased over that of oxygen consumption on the operative side. This slight increase may be considered as a factor of the method, rather than impairment of segmental function.

In the group with bilateral surgery, the parallel increase of percentage M.B.C. with the increase of number of remaining segments is similar to that of

TABLE I. VENTILATION—POSTSEGMENTAL RESECTION

PATIENT	AGE	SURGERY	NO. REMAINING SEGMENTS	INTERVAL YR.	INTERVAL MO.	M.B.C. L./MIN.	M.B.C. PRED. %	TIMED V.C. (%) 1 SEC.	2 SEC.	3 SEC.	WALKING VENT. (L./MIN.)	W.V. M.B.C.	A.V.I.	REMARKS
						Unilateral								
F. M.	40	L basal, lingula	13	2	3	56	69	80	88	94	11.9	0.17	0.84	Has bilateral bronchiectasis
M. M.	23	RML	16	2	5	145	92	90	95	98	21.0	0.15	0.93	Obliterated fissures, segmental technique used
E. C.	30	LLL lingula	12	0	9	68	98	75	94	100	17.1	0.25	0.98	
L. R.	47	Lingula, sup. seg. LLL	15	1	6	66	88	78	92	96	13.9	0.21	0.98	
N. F.	53	Post. & lat., basal LLL	16	0	7	88	74	82	90	98	22.5	0.25	0.93	
A. D.	29	LLL lingula & ant. LUL	11	0	2½	84	67	80	90	95	18	0.21	0.90	Left temporary phrenic
						Bilateral								
G. Z.	29	L basal, R basal	11	1	7	104	83	76	94	98	16.9	0.16	1.10	
A. R.	27	RML, RLL, L basal	8	0	7	80	140	70	85	90	25.0	0.31	1.0	
F. O.	43	LLL, lingula, RML & rt. basal	6	1	6	67	55	--	--	--	13.8	0.21	0.59	
H. K.	29	L basal & lingula, R basal & RML	7	1	11	55	58	78	85	100	21.5	0.39	0.84	
R. H.	28	LLL, lingula, RML	10	2	0	75.2	91	80	85	95	19.7	0.26	1.79	
C. F.	34	LLL, lingula, RML & ant. RUL	9	3	1	73	81	--	--	--	12.8	0.17	1.16	
H. M.	33	RML, lateral basal RLL, L basal, inf. lingula	11	3	2	56.8	78	76	91	95	17.0	0.30	0.94	
R. D.	39	LLL, lingula, R basal, inf. subseg. of ant. seg. of RUL, RML	5½	-	-	39	53	90	95	100	12.2	0.31	1.2	

TABLE II. LUNG VOLUMES—POSTSEGMENTAL RESECTION

PATIENT	REST RESP. RATE /MIN.	TIDAL VOL. (C.C.)	MIN. VOL. (L./MIN.)	O_2 UPTAKE (C.C./MIN.)	VENT. EQUIV. (L./MIN.)	VITAL CAP. (C.C.)	INSP. CAP. (C.C.)	EXP. RES. (C.C.)	FUNCT. RESID. CAP. (C.C.)	RESID. CAP. (C.C.)	T. LUNG CAP. (C.C.)	RV/TC %	MIXING INDEX $\%N_2$
Unilateral													
F. M.	7	746	5.2	208	2.6	2274	1156	888	----	----	----	--	- --
M. M.	12	723	8.3	448	1.86	4438	2932	1527	1645	118	4556	3	0.77
E. C.	22	376	8.4	325	3.2	1799	1657	326	917	591	2390	25	0.55
L. R.	11	806	8.8	314	2.8	2120	1640	393	----	----	----	--	- --
N. F.	20	668	13.6	453	3.2	3076	2307	789	1843	1054	4130	25	1.13
A. D.	14	748	10.4	369	2.8	2932	2265	689	1914	1044	3976	26	1.20
Bilateral													
G. Z.	12	607	7.6	326	2.4	3107	1893	1174	2215	1041	4148	25	0.67
A. R.	20	397	7.9	334	2.4	2883	1722	1019	2759	1740	4623	38	2.46
F. O.	--	630	6.3	270	2.3	3760	2290	1060	----	2470	5900	42	- --
H. K.	12	728	8.7	304	2.5	2023	1376	749	1584	835	2858	29	1.03
R. H.	22	346	7.6	256	3.0	1496	916	631	1150	519	2015	26	1.37
C. F.	--	445	5.3	179	3.1	2139	1116	883	----	----	----	--	- --
H. M.	17	446	7.5	248	4.4	2393	1521	1014	1763	749	3142	24	1.15
R. D.	15	348	5.2	241	1.9	1267	879	552	1322	666	1933	35	1.11

TABLE III. DIFFERENTIAL BRONCHOSPIROMETRY AND ARTERIAL BLOOD GASES—POSTSEGMENTAL RESECTION

		RIGHT LUNG					LEFT LUNG					RESTING, ARTERIAL BLOOD GASES			
PATIENT	TOTAL SEG.	SEG.	O_2%	VENT. %	V.C. %	V. EQ. (L./MIN.)	SEG.	O_2%	VENT. %	V.C. %	V. EQ. (L./MIN.)	O_2 SAT. %	pH_a 37° C.	pCO_2 (MM. HG.)	TOTAL CO_2 (MM./L.)
Unilateral															
F. M.	13	10	59.6	58.5	63.9	4.2	3	40.4	41.5	36.1	4.3	---	---	---	---
M. M.	16	8	45.8	52.7	49.9	2.2	8	54.2	47.3	50.1	1.7	95	7.53	33	27
E. C.	12	10	81.2	73.8	77.3	2.1	2	18.8	26.2	32.7	3.2	94.6	7.45	33.5	23.2
L. R.	15	10	70	58.1	66.2	2.16	5	30	41.9	33.8	3.65	97	7.25	48.8	21.3
N. F.	16	10	63.4	59.1	68.8	4.64	6	36.6	40.9	31.2	5.56	94	7.38	49	23.4
A. D.	11	10	77.4	72	80	2.4	1	22.6	28	20	3.2	95	7.45	39	26.7
Bilateral															
G. Z.	11	6	47	54	55	5.47	5	53	46	45	4.1	96	7.41	40.5	25.4
A. R.	8	3	39.3	38	45.7	3.9	5	60.7	62	54.3	1.5	97	7.41	30	19.5
F. O.	6	4	59.5	66.4	62.9	5.6	2	40.5	33.6	37.1	4.2	---	---	---	---
H. K.	7	4	58.2	67.3	60	3.1	3	41.8	32.7	40	2.1	95	7.53	33	27
R. H.	10	8	66.7	71	72	4.06	2	33.3	29	28	3.03	96	7.36	45.5	25.5
C. F.	9	7	76.5	47.6	62	1.9	2	23.5	52.4	38	5.8	---	---	---	---
H. M.	11	7	58	48	51	2.8	4	42	52	49	4.2	92	7.55	29	25
R. D.	5½	3½	---	---	---	---	2	---	---	---	---	---	---	---	---

the unilateral group. Mild clinical dyspnea was evident in only one case (A.R.) following the exercise test. This patient was the only one of the group who had a timed vital capacity of 70 per cent in one second (normal value 75 per cent). The oxygen consumption is greater in the lung that has the greater number of segments (Table III). In five of the seven cases, on the side with the least number of remaining segments, the ventilation is less than the percentage of oxygen consumption.

Arterial blood gases were obtained in ten of the fourteen patients. The arterial oxygen saturation, pH_5, pCO_2, and total CO_2 were within normal limits on all of the patients studied (Table III).

Total lung volumes were obtained on eleven of the fourteen patients and the results are listed in Table II. In the unilateral surgical group all of the values were within normal limits. In the group with bilateral surgery, four patients had an increase in residual volume. Only one patient of the entire group had an elevated alveolar nitrogen value after seven minutes of oxygen breathing.

DISCUSSION

Clinical.—Prior to surgery, none of the six patients with unilateral surgery were incapacitated so that they could not perform their normal, everyday activities. Following surgery, they did not notice any remarkable change in their clinical breathing reserve or exercise tolerance, but did note a disappearance of symptoms.

Patient F. M. in the unilateral surgery group did have bilateral bronchiectasis but only the diseased segments on the left (basal segments and lingula) were resected. The symptomatic improvement was so marked that surgery on the right was deferred. The differential study indicates that the three segments on the left side now contribute approximately 40 per cent to the total oxygen consumption and ventilation as compared to the ten segments on the right. Of the ten segments in the right lung, six of these are bronchiectatic and presumably are not contributing much to the patient's pulmonary function.

Patient L. R. had frequent, severe pulmonary hemorrhages, and bronchography demonstrated cystic bronchiectasis of the lingula and the superior segment of the left lower lobe. If the unit of excision had been lobe instead of segment, it would have been necessary to excise the entire left lung to do away with this patient's symptoms. She now has fifteen segments remaining in the two lungs, with ten on the right and five on the left, and a differential study indicates that the remaining five segments on the left contribute about one-third of the total function.

Of the eight patients with bilateral bronchiectasis, only two were able to carry on any resemblance of normal activity prior to surgery. Clinically, they were short of breath, complained of cough, excessive, purulent sputum, and, in several cases, severe pulmonary hemorrhages. They have noted marked improvement in their exercise tolerance and breathing reserve, and also a disappearance of the symptoms since surgery.

Patient A. R. had extensive saccular bronchiectasis involving the right middle lobe, right lower lobe, and basal segments of the left lower lobe. He had been unable to attend school or to work because of the severe symptoms. Since surgery, this patient now works an eight-hour day, climbs two flights of stairs without dyspnea, and is free of cough and sputum.

Patient F. O. is a 43-year-old priest who now has only six segments, or one-third of the total number in normal individuals. Four of these are on the right and two on the left and postoperative bronchograms are normal. Differential studies showed normal distribution of oxygen consumption, ventilation, and vital capacity. His M.B.C. is 66.9 liters per minute, ventilatory equivalent is 2.36 liters, and he does have a residual volume of 42 per cent of his total capacity, indicating some pulmonary emphysema or overdistention. This degree of emphysema has not caused clinical dyspnea, and this patient can perform his required activities now for the first time in many years.

Patient C. F. had extensive bronchiectasis involving the left lower lobe, lingula, right middle lobe, and the anterior segment of the right upper lobe. The left lower lobe was removed in December, 1943, the lingula in 1947, and the involved segments on the right in 1948. She now has only 50 per cent of her total number.

Pulmonary function studies were first done in 1947 (prior to lingulectomy) and showed a M.B.C. of 74 L. per minute or 80 per cent predicted, walking ventilation of 12.5 L. per minute, and ventilation equivalent of 2.9 L. per minute. These studies were repeated following a lingulectomy, right middle lobectomy, and resection of anterior segment of the right upper lobe. It can be seen that removal of the additional segments (three on right, two on left) did not materially impair the mechanism of ventilation as shown by the M.B.C. The functional relationships between the right and left lung were also not altered. This case clearly demonstrates the beneficial effects of segmental surgery; namely, (1) eradication of diseased segments plus symptoms, and (2) conservation of normal pulmonary tissue and function.

Physiologic.—Preoperative pulmonary function studies were not obtained in this series for the following reasons:

1. The presence of abnormal amounts of secretions in the bronchial system obstructs air flow in bronchi of normal segments and causes an increase in ventilatory effort.

2. Excessive secretion impedes oxygen consumption.

3. In unilateral cases, the normal lung compensates for the altered function of the involved side.

Postoperative pulmonary function studies in the unilateral and bilateral groups indicate that segmental pulmonary resection does conserve the integrity of a large number of factors related to ventilation. A comparison of the determined M.B.C. to predicted value is recorded in Fig. 5. As there is an increase in the number of healthy segments, the determined M.B.C. approaches the predicted value for normal. This only emphasizes the im-

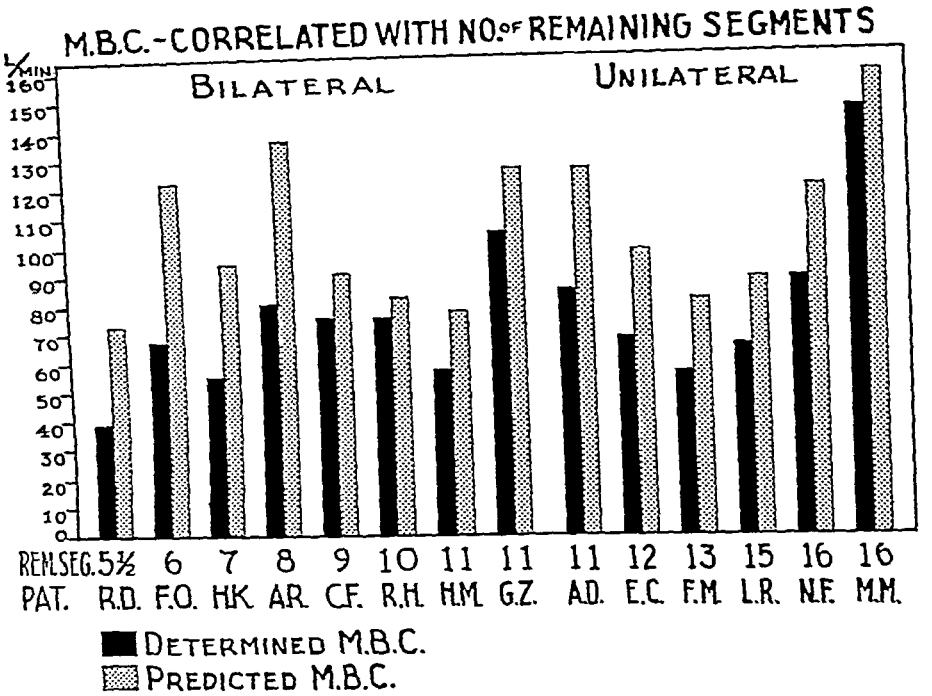


Fig. 5.—The bilateral group includes Patients R. D. to G. Z.; the unilateral, Patients A. D to M. M.

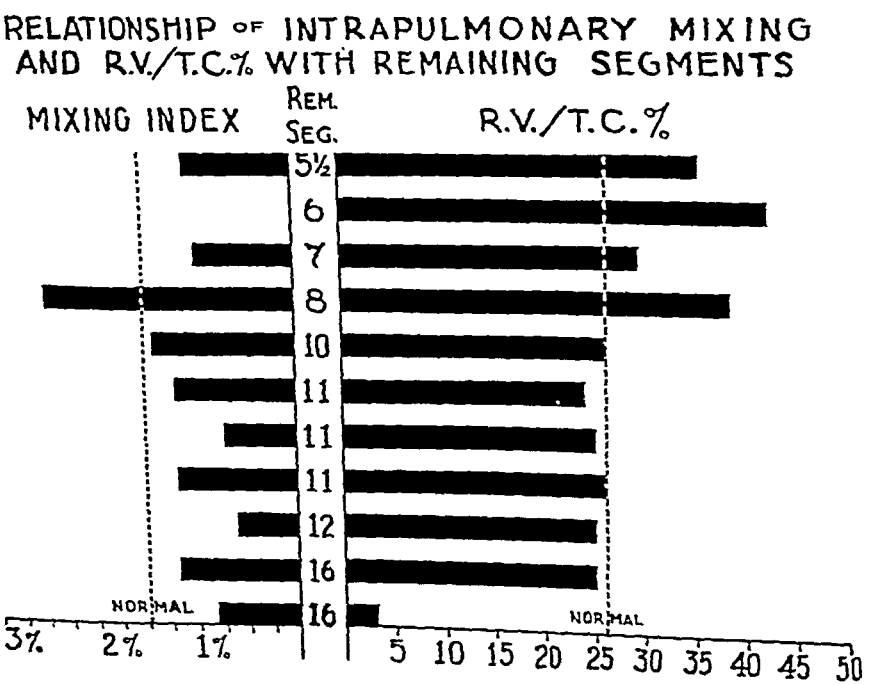


Fig. 6.

portance of the segments as the unit of excision in treating bronchiectasis. The determined M.B.C. is low in some of the patients but these values do not correlate with their clinical respiratory reserve. This supports Comroe's[12] contention that reduction in M.B.C. must be large to be significant because there can be individual deviation of 25 to 35 per cent of mean group values. Each conserved segment contributes to oxygen consumption and ventilation. This is well demonstrated in patient A.D. The one remaining segment on the left side participates in 21 per cent of the total oxygen consumption. The comparison of percentage oxygen consumption to percentage ventilation of the conserved pulmonary segments demonstrates efficient function. In the bilateral group, the side that had the greater number of segments removed ventilated within normal limits.

The four patients with an increased residual volume had the least number of remaining segments—5½, 6, 7, and 8 (Fig. 6). These findings are consistent with overdistention and most likely represent hyperinflation rather than true emphysema. The normal alveolar nitrogen values represent adequate intrapulmonary mixing of gases (Fig. 6). This supports the belief that hyperinflation does not cause pulmonary disability.

Dissection of the diseased pulmonary segments with a minimum of operative manipulation of the healthy segments, plus a minimum of postoperative respiratory complications, are important etiological factors in preserving normal function in the remaining lung tissue.

CONCLUSION

The pulmonary function studies in this series, following unilateral and bilateral segmental resection for bronchiectasis, demonstrate the necessity of saving normal lung segments.

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DISCUSSION

DR. J. GORDON SCANNELL, Boston.—I should like to confine my remarks to one aspect of Dr. Overholt's excellent presentation. It has occurred to us that one may question the propriety of a simultaneous bilateral resection on other than technical grounds. It has been both my training and my experience that in bronchiectasis one should be guided more by the demonstrated liability of a segment to a patient rather than by the radiologic diagnosis. We have frequently found that in a proposed bilateral program we have done one side and found it unnecessary to do the second side. Particularly has this been true in the scattered, radiologically involved segments which, in fact, may be merely scars of disease and may actually be doing some good in a patient's chest by occupying space.

DR. C. E. O'BRIEN, Ann Arbor.—The matter of bilateral pulmonary resection has raised certain problems which have been of considerable interest to us. There is, first, the question of how much pulmonary tissue may be removed allowing the operation to be compatible with life and with a reasonable degree of activity. Second, there is the matter of what happens to the remaining pulmonary tissue, and one thinks of the problem of overdistention as well as the pulmonary vascular resistance which may develop, with its effect on the function of the right heart. Third, we have been interested in the matter of the optimum time for operation upon the second side, and I am much interested to hear the idea proposed of simultaneous operations.

Our series is a long-range study, of course, but thus far our cases have demonstrated certain findings rather consistently. First, each patient who has had bilateral resection has demonstrated some degree of impairment of ventilatory function, diminution of vital capacity, and maximum breathing capacity. Second, each patient has shown some overdistention of the remaining pulmonary tissue as evidenced by the *increase* in the ratio of residual air to the total capacity. Third, there has been a tendency toward a low resting arterial oxygen saturation which is accentuated by exercise. And last, there have been changes in the ventilation equivalent suggesting that the function of the remaining pulmonary tissue is somewhat less efficient than normal.

I have a few slides which I thought might illustrate one or two things we have observed: (Slide.) This is a young woman 20 years of age who had thirteen segments removed, having had the lingula of the left upper and the left lower and also the right middle and right lower lobes removed. You will notice that there is a diminution in ventilatory function and there is some increase in residual air. I think probably the latter is ominous from the point of view of what is going to happen to this young woman in years to come. You will notice that while there is slight evidence of arterial oxygen desaturation, it is not striking. This young woman lives a normal life; she works full time and enjoys dancing and bowling and such activities.

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(Slide.) On the other hand, this patient is 37 years of age, with both lower lobes removed. This illustrates, we believe, certain important principles. The man was recognized to have, in addition to bilateral lower lobe disease, a minimal amount of bronchiectasis in the anterior segment of the left upper lobe. It was thought to be minimal, but when that segment of the left upper lobe lost the advantage of its position so far as adequate drainage is concerned, he began to have much more severe symptoms from that bronchiectasis which was left behind. You will note that there is a relatively severe degree

of impairment, particularly in the maximum breathing capacity, less in the vital capacity; the intrapulmonary mixing is very poor, and you will notice the great degree to which this man desaturates the arterial blood on exercise. We wonder about the point at which the function is lost in patients such as this. I might say that this man suffered a complicated postoperative period with bronchial fistula on the left side which prolonged the convalescence and resulted in certain pleural changes, which we think are responsible for some of this impairment of function, as is the residual disease.

(Slide.) So to get at the basis of this we thought that it would be interesting to know how the remaining pulmonary tissue on the side of resection functions immediately after operation. These are observations which were made immediately after operation with the patient still under anesthesia. We were surprised to notice the relatively good function of the remaining pulmonary tissue. In some cases the oxygen uptake was actually increased, in other cases in which abnormal tissue was removed the ventilation equivalent was improved, and in general it may be said that the immediate postoperative function, so far as can be determined, is surprisingly good. We think this emphasizes the point which has been mentioned by others, namely, that what happens to the pleura because of complications in the postoperative period is of exceedingly great importance in the final functional picture.

In summary, I would like to say that I am in complete agreement with the principle of preserving all normal pulmonary tissue. I think this is important from the point of over-all function obtained from the segments which are preserved, and it is also important from the point of view of taking up the space which would have to be filled by overdistention of the remaining segments, if normal segments were removed simply for the purpose of doing a total lobectomy rather than segmental resection. Second, one must beware of bronchiectasis which appears to be of little significance initially, which is in a favorable position for bronchial drainage from the upper lobes, but which loses this favorable position and becomes dependent and symptom-producing when it serves to fill the space it must occupy after lower lobe resection. Third, I will reiterate that early postoperative pulmonary function is surprisingly good in the segments remaining on the side of operation, and one must bend every effort to prevent postoperative pleural complications in an effort to preserve this function.

DR. OSLER ABBOTT, Atlanta.—I should like to mention one factor in regard to resection and the loss of a major amount of lung tissue in the presence of a large amount of dead space. It is a very simple matter to make a suture purse string in the diaphragm and insert a catheter into the peritoneal cavity, creating a pneumoperitoneum just before closing the chest. His comfort, postoperatively, is increased; his diaphragmatic function and his temporary pulmonary disturbance from overdistention of the lung will be greatly aided.

DR. OVERHOLT (Closing.).—Dr. Scannell has brought up a very important point about the evaluation for the need of doing the second side in bronchiectasis. It happens that we have seen some 220 patients with bilateral bronchiectasis and elected to do bilateral surgery in only seventy cases. I agree that it should be clearly evident that bilateral resection will be required before such treatment is planned. Clinical evaluation, character of the bronchial dilatations, and bronchoscopic study may clearly indicate that the extent of the septic reservoir on both sides requires removal. It is then possible to tell in the beginning that a particular patient may or may not need to have both those septic reservoirs removed. In the selection of cases or in proposing surgical treatment, every surgeon must place himself in the position of the patient and do for him what he thinks he would like to have done for himself. In the particular cases we selected for bilateral resection, it was decided that if I were in the patient's place, I would like to have my problem managed in that particular way. I should point out, however, that probably the number of cases that may be handled that way are very few, and each surgeon has to make his own decision and work out his own solution for the problem that will provide

the greatest factor of safety. We did it in order to eliminate the spillage from the contralateral side.

It might have been interesting if we could have had preoperative function studies made in all of the cases included in this report. There were four reasons why we did not have these studies: first, the presence of abnormal quantities of secretion in the bronchial system obstruct air flow in normal segments and cause an increase in ventilatory effort; second, excessive secretion impedes oxygen consumption; third, in the unilateral cases, the normal lung compensates for altered function of the involved side; fourth, at the time most of these studies were done, we did not have the physiology laboratory setup to do the studies—which fact alone provides an adequate reason for not having had preoperative studies in these particular patients.

Dr. O'Brien, in his very excellent discussion, brought out an interesting point. That is, that in the very early studies of function, immediately after partial lung resection, function as measured may be extremely good and that possibly some of the late disturbances in function are most likely due to the effect of pleural constriction. This emphasizes the great need for careful attention in the postoperative period to immediate reexpansion of remaining segments and removal of air and fluid from the pleural space.

It might be of interest to interpose a brief comment about other technical matters:

1. In bilateral cases, which side should be treated first? It has been our practice to remove the disease in the side which constitutes the greater burden. If it can be determined by symptoms, x-ray examination or bronchoscopy which side is acting as the principal reservoir for the collection, concentration, and stagnation of secretions, those segments should receive first attention. In a significant number of cases, all symptoms or constitutional effects of the disease disappear or are so minimal that excisional therapy on the side of lesser disease may be indefinitely postponed.

2. It has been our observation that the postoperative management of patients may be complicated after the first single-sided operation. One is confronted with problems of reexpansion on one side while the remaining segments are bathed in secretions from the contralateral septic reservoir through the intercommunicating bronchial system. To solve the problem of the flooding of the remaining healthy segments with material from the contralateral lung in bilateral disease, we have, for some time, toyed with the idea of completing the excision of all abnormal segments, even if bilaterally situated, in one stage.

3. From a group of seventy patients in whom a bilateral operation has been completed, in two instances a one-stage bilateral operation was done. In one other patient (nonbronchiectatic), who suffered from bilateral disease which required excisional therapy, the treatment was completed in one stage.*

(Slide 1.) A 46-year-old woman suffered from bronchiectasis in the inferior subsegment of both anterior segments, the right middle lobe, and the lingula. With the patient in the prone position, the diseased segments on the right side were removed on Jan. 8, 1952. While the wound was being closed, a left thoracotomy was made and the involved segments on that side were removed. The patient's convalescence was prolonged because of the necessity to drain a small localized empyema which developed in an anterior location. Offending organisms were resistant to antimicrobial agents. The hospital stay extended over a period of twenty-six days.

(Slide 2.) A 29-year-old woman had a ten-year history with bronchiectasis in the right middle lobe, and the left basal group, and lingula. On Jan. 26, 1952, the left side was treated first and since every detail of the procedure was expeditiously executed, it was deemed safe and advantageous to resect the middle lobe immediately, and this was done. The convalescence was uneventful, and the patient was discharged on the thirteenth postoperative day.

*Overholt, Richard H., Rheinlander, Harold F., and Bolton, Houck E.: Simultaneous Bilateral Pulmonary Segmental Resection, Bull. New England M. Center 14: 1, 1952.

(Slide 3.) A 53-year-old man had a survey lesion. An abnormal shadow suggestive of a primary tumor was found in the right upper lobe and a discrete "coin" shadow in the left upper lobe. On Feb. 7, 1952, a right thoracotomy revealed a mass in the contracted posterior segment and, after its removal, a tuberculous lesion was found. While the wound was being closed, a second team opened the left chest where a discrete hamartoma was found which was removed by enucleation. Convalescence was uneventful, and the patient was discharged on the tenth postoperative day.

None of the three patients required tracheal or bronchoscopic aspiration postoperatively. Re-expansion was satisfactory in all cases. None experienced more than the average amount of discomfort. There was no appreciable splinting of the thoracic cage nor was there dyspnea.

THE INDICATIONS FOR AND THE RESULTS OF COMMISSUROTOMY FOR MITRAL STENOSIS

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SELDOM in the history of the medical profession has the introduction of a single surgical procedure so revolutionized the therapeutic demeanor and prognostic thinking toward a common disease entity as has commissurotomy for mitral stenosis. In five short years of pioneer effort, the physical and mental dissolution of the patient suffering from this disastrous and cicatricial end result of rheumatic fever has been revitalized and remodeled to an attitude of hope and assurance. Indeed, the demonstration that the left atrium of the heart can be entered at will to reconstruct surgically the obstructed mitral valve has paved the way for similar approaches to each of the other valves and has led to the establishment of intracardiac surgery as a definitive specialty.

Three years ago, before this association, our initial efforts in the field of valvular surgery were reported.[1] Much skepticism was engendered in the minds of both protagonists and antagonists. The fact that in years gone by a number of attempts to overcome mitral obstruction had terminated fatally in the hands of highly competent surgeons tended to dull and becloud the reception of a possible repeat performance. The demonstration, however, that the release of obstructed valves could be accomplished without the simultaneous production of lethal or even significant valvular insufficiency—originally considered to be the inevitable corollary to such surgery—restored to reality the hopes and dreams of many investigators both living and dead whose original efforts had until recently remained unheralded.

It is the purpose of this article to present the complete data pertaining to our first 256 consecutive commissurotomies for mitral stenosis. Previous communications have reviewed the contributions of the many investigations in this field.[2-9] These reports have also detailed the pathologic and physiologic considerations of this valvular deformity, together with the technique employed for its surgical correction.[10-15] It is, therefore, sufficient to state here that mitral commissurotomy is a procedure in which the individual anatomic leaflets of the stenotic valve are separated. By incising the angles or commissures of the mitral slit its obstructing effect can be overcome and a considerable degree of valve function can be re-established. It is to be

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noted that no valve tissue is removed, thus allowing the liberated though thickened and deformed valve leaflets to open during ventricular diastole and approximate during ventricular systole without the production of additional significant regurgitation (Fig. 1).



A. *B.*

Fig. 1.—View of the stenotic mitral valve cone from the left ventricle. *A*, Right index finger and Glover-Avery guillotine knife in position for incision of anterolateral commissure. *B*, Anterolateral commissure completely separated greatly enlarging the orifice with partial restoration of valve function.

ANALYSIS OF CASES

We have performed 256 mitral commissurotomies from Feb. 2, 1949, to April 30, 1952. Fifty-three of these have been performed since Jan. 1, 1952, and except for mortality data are not included in the statistical analysis at this time. Similarly, thirty-nine others were performed for and with other thoracic surgeons on their own services throughout the country by invitation. These will be reported in detail by them at some future time. The operative mortality rate for the entire series was 5.4 per cent, and the over-all mortality, including late deaths, was 7.8 per cent (Fig. 2). These later deaths occurred almost exclusively in stage V cases, over six months after surgery, from a continuation of their irreversible cardiovalvular and pulmonary vascular disease unaffected by surgical intervention.

The body of this report concerns itself with the remaining 164 consecutive cases with a six-month to three and one-half year follow-up period for analysis. A lapse of at least six months is essential before results may be evaluated, for experience has shown that in many instances at least this period of time must pass before the patient can fully appreciate the full subjective benefits of surgery. Actually, a longer period is often required before all objective evidences have become stabilized.

Of these 164 cases, 118 were female patients and 46 were male. The youngest was 4 years of age and the oldest was 59, with an average age of 35 years. Most of these patients were far advanced in the course of their disease and represented the late or terminal stages of mitral stenosis. They have been grouped according to our previously published classification as shown in Table I.

TABLE I. STAGES OF MITRAL STENOSIS

STAGE	NUMBER	DEATHS		MORTALITY %	
		OPERATIVE	LATE	OPERATIVE	LATE
I. Asymptomatic	0	0	0	0	0
II. Statically incapacitating	14	0	0	0	0
III. Progressively incapacitating	105	5	1	4.7	.9
IV. Terminally incapacitating	32	3	0	9.3	0
V. Irreversible	13	2	8	15.3	61.5
Total	164	10	9	6.0	5.4
Over-all Mortality		11.5%			

This classification has been prepared in an effort to combine a functional and therapeutic yardstick for evaluation of the patients for surgery. *Stage I* includes those with the auscultatory findings of mitral stenosis but who as yet have no symptoms. Cases in *stage II* have progressed to the point where symptoms under physical activity have developed, but the patient, living within his

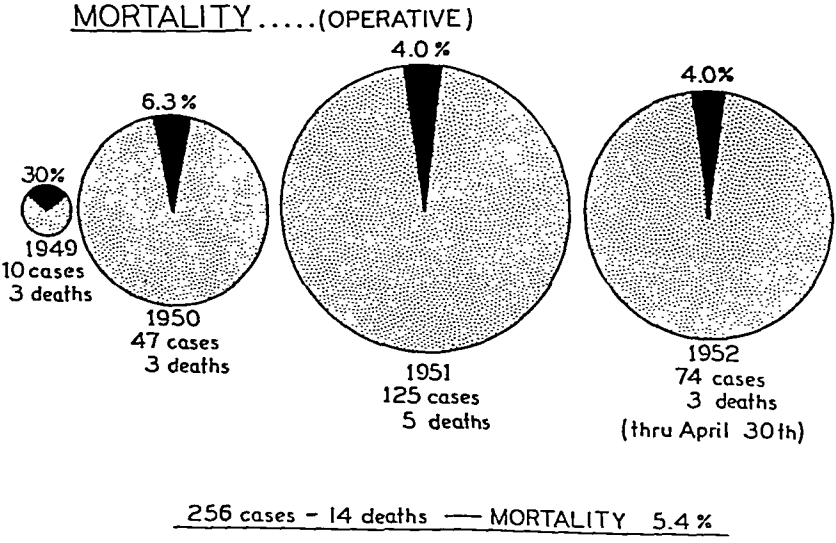


Fig. 2.—Mitral commissurotomy, showing mortality statistics by years from Feb. 2, 1949, through April 30, 1952.

own limitations, remains on an even plateau. *Stage III*, the largest group, and one encompassing many variables, takes in those who, despite the best medical therapy, are losing ground. *Stage IV*, terminally incapacitating, is made up of those patients in whom there is constant evidence of congestive failure even with limited physical activity. Most of these can be rendered relatively free

of their accumulating tissue fluid *only* by the strictest of medical regimens. A certain small percentage of those in this group will ultimately prove after surgery to have been in *stage V* and to have had irreversible changes. As yet, it has been impossible to separate routinely the patients in these two stages by clinical and physiologic methods; hence, we reserve stage V to classify those whose condition, despite a technically adequate commissurotomy, remains relatively unchanged.

Status Prior to Admission.—Progressive dyspnea and fatigue were present in varying degree in each of the 164 cases (Table II). In addition to this evidence of cardiopulmonary disability, 110 patients had already progressed to the stage of right heart (congestive) failure as evidenced by one or more episodes of hepatomegaly, ascites, and peripheral edema; 59 had had one or more attacks of frank hemoptysis, in several instances to an almost exsanguinating degree. This does not include the many who had expectorated frothy material during bouts of pulmonary edema.

TABLE II. STATUS PRIOR TO ADMISSION

Progressive dyspnea and fatigue	164	100.0%
Congestive heart failure	110	67.1%
Hemoptysis	59	35.9%
Arterial embolism	21	12.8%

In twenty-one patients, cerebral, visceral, or peripheral emboli had occurred on one or more occasions. Of these, fourteen had auricular fibrillation, seven had normal sinus rhythm, and all but six had a history of one or more attacks of congestive failure. It is of considerable interest that only nine of these had gross thrombi in the left auricular appendage at operation. In one additional case the pathologist was able to report the presence of a small thrombus deep in the trabeculae of the excised appendage which was not noted during the commissurotomy. Of greatest significance is the fact that no recurrent embolic episodes have occurred in any of these twenty-one cases (longest three years). It has been previously emphasized that ligation of the base of the left auricular appendage was responsible for this result. However, of equal if not greater importance would seem to be the relief of left atrial stasis accomplished by opening the valve.

TABLE III. ADMISSION FINDINGS

"Pure" mitral stenosis	97	
Mitral stenosis with insufficiency	42	
Mitral stenosis with insufficiency and aortic insufficiency	11	
Mitral stenosis with aortic insufficiency	14	
Left ventricular enlargement (x-ray) (large normal to minimal)	24	
Auricular fibrillation	69	(42%)
Congestive heart failure	43	(26%)
Electrocardiogram: RAD	140	
NAD	23	
LAD	1	

Status Upon Admission.—In the initial phase of this surgical program, every attempt was made to select cases of relatively pure mitral stenosis to insure a proper base line for evaluation of the surgically treated stenotic valve alone (Table III). It has since become apparent that the auscultatory findings of a minimal to moderate systolic mitral and/or aortic systolic or diastolic murmurs in addition to the classical mitral diastolic murmur may be of little consequence provided mitral *stenosis* is the *predominant factor* in the production of the patient's disabling symptoms. Ninety-seven were classified as "pure" mitral stenosis when no regurgitant jet could be felt by the exploring finger at the time of surgery. Prior to this, on purely auscultatory grounds, at least one-third of these had been thought to have associated mitral insufficiency which could not be corroborated at the operating table. It is therefore felt that, contrary to classical teaching, *pure* mitral stenosis can and, not infrequently, does occur without accompanying regurgitation. In forty-two cases, there was an associated minimal (grade 2 systolic murmur) mitral insufficiency, and in seven others there was aortic insufficiency of minimal degree. In fourteen individuals a mitral diastolic rumble and an aortic diastolic murmur were the only murmurs heard. Four others were also complicated by the presence of an aortic systolic murmur of minimal degree.

In the selection of cases for surgery, it is obvious that the careful auscultatory examination by an experienced cardiologist is paramount. The recognition of a typically uncomplicated mitral stenosis by stethoscopic methods presents no problem. The difficulty does not lie with this type of individual but rather with the patient who presents evidence of coexisting mitral stenosis and insufficiency, or multivalvular disease. Here, one must decide whether the mitral stenosis is the predominant lesion. Expert fluoroscopic and radiologic evaluation, with emphasis on individual chamber enlargement, can be of inestimable value in this respect.

One hundred forty of the cases presented a normal left ventricle, and in twenty-four there seemed to be minimal enlargement. In these twenty-four, mitral insufficiency or associated aortic valvular disease was *dynamically* expressing its presence by enlargement of the left ventricle, a finding which we have come to regard as evidence tending to contraindicate surgery. Slight enlargement of the left ventricle, due to mitral insufficiency, is not an absolute contraindication but suggests the possibility of a more equivocal surgical result. If left ventricular enlargement is due to an aortic valvular lesion, commissurotomy might be questioned at this time, although recently two cases of pure mitral and aortic stenosis in which both valves were opened at the same operation have been treated very successfully. The recognition of minor degrees of left ventricular enlargement in the presence of a markedly enlarged right ventricle may well be perplexing. Enlargement of all chambers of the heart is an absolute contraindication.

Auricular fibrillation is not a contraindication to surgery per se unless the ventricular rate is entirely uncontrollable. Sixty-nine patients had auricular fibrillation, and the eventual results have compared quite favorably with those who had sinus rhythm.

Forty-three patients presented themselves in various stages of congestive heart failure, although many others were known to have been in this state prior to admission. In all but thirteen this failure was well controlled before surgery, wherein there existed many years of chronic failure. Except for minimal clinical evidences, these too eventually responded. The period of medical preparation of patients in this status was more prolonged, and in several instances six weeks or more were required. Some of the more dramatic results were achieved in this group after a prolonged period of convalescence.

The importance of hypertension of the pulmonary circulation in the natural history of mitral stenosis has been appreciated for many years. Such classic symptoms as dyspnea, orthopnea, and hemoptysis have been thought to be indications of increased pressure in the pulmonary vascular bed. The roentgenologist and pathologist have supported the clinician's contention of the importance of pulmonary hypertension by describing hypertrophy of the right ventricle, marked enlargement of the left atrium and pulmonary artery, and sclerotic changes in the large and small pulmonary arteries.

In patients with mitral stenosis, right heart catheterizations have recorded high systolic pressures in the right ventricle. Borden and his associates[16] found a rough correlation between the degree of pulmonary artery hypertension and the clinical manifestations. Comparable findings and correlations were observed in forty-five of our first fifty patients in whom catheterization of the right heart was done. The upper limit of the pulmonary artery pressure found in this laboratory in normal subjects is 30/10 mm. Hg. As might be expected, the higher the pressure in the pulmonary artery, the closer the correlation with the clinical and roentgen findings. Usually a systolic pulmonary artery pressure of 50 mm. Hg or greater was associated with a history of prolonged functional disability, episodes of acute pulmonary congestion, hemoptysis, or congestive failure. Such pressures, also, were commonly associated with fluoroscopic and x-ray evidence of marked left atrial and right ventricular enlargement and dilated pulmonary arteries. Electrocardiograms in 140 of this group showed right axis deviation (plus 90 degrees or more), thus reflecting somewhat the degree of pulmonary hypertension.

In a previous communication,[17] the early results of commissurotomy upon the pulmonary arterial pressure have been presented in great detail. In general, it may be stated that the higher the preoperative pressure the greater the postoperative drop, noted particularly in those above 80 mm. Hg systolic in the pulmonary artery. Those ranging between 40 and 80 mm. Hg systolic naturally dropped correspondingly less. The over-all average drop in right heart and pulmonary arterial pressures within one month of surgery was 15 mm. Hg systolic (extremes 0 to 40).

Of much greater interest and significance are the pressure changes which developed and stabilized over many months. One can appreciate the difficulties encountered in obtaining long-range recatheterization data. Although most will and have returned from great distances for postoperative clinical evaluation, few are interested in submitting to a third catheterization (preoperative, early postoperative, and late postoperative). Nevertheless, such

TABLE IV. COMMISSUROTOMY—PRESSURE CHANGES
(mm. Hg—Lilly Manometer)

CASE AND DATE OF OPERATION	PREOPERATIVE		POSTOPERATIVE				MONTHS	RESULTS
			INTERVAL		MOST RECENT			
	RT. VENT.	PUL. ART.	RT. VENT.	PUL. ART.	RT. VENT.	PUL. ART.		
1. J. H. 6/2/50	67/-2 (21)	78/24 (42)	31/4 (13) 1 mo.	30/16 (5)	77/11 (33)	75/29 (44)	5	Unimproved
2. A. N. 10/18/50	(37)	(72)			29/4 (12)	27/14 (18)	9	Excellent
3. G. K. 5/19/50	50/2 (18)	48/24 (32)	31/-2 (9) 1 mo.	36/12 (20)	47/4 (18)	52/23 (33)	10	Improved
4. G. D. 7/6/50	90/0 (30)	84/40 (55)	108/9 (42) 1 mo.	93/48 (63)	50/-2 (19)	50/18 (29)	10	Excellent
5. J. A. 11/8/50	57/-15 (39)	57/24 (35)			45/5 (18)	40/19 (26)	12	Excellent
6. E. M. 1/23/50	55/36 (42)	49/30 (36)	44/0 (15) 1 mo.	45/22 (30)	28/0 (9)	35/10 (18)	13	Improved
7. E. I. 11/9/50	93/6 (35)	105/24 (51)			31/3 (12)	38/13 (21)	14	Excellent
8. M. D. 1/5/51	32/-2 (13)	30/16 (21)			20/8 (12)	20/6 (11)	14	Excellent
9. E. C. 11/3/50	64/-3 (25)	75/27 (43)			30/0 (10)	30/10 (17)	14	Excellent
10. V. S. 6/27/49	141/5 (50)	143/56 (85)	46/-2 (18) 4 mo.	44/13 (23)	33/14 (20)	36/15 (22)	15	Excellent
11. F. L. 7/27/50	68/0 (23)	-	89/8 (35) 7 mo.	89/39 (56)	52/5 (21)	-	16	Excellent
12. J. B. 2/2/49	90/17 (41)	-	45/0 (15) 2 mo.	50/16 (27)	48/3 (18)	-	20	Excellent
13. V. S. 4/11/50	100/9 (39)	130/59 (83)	90/2 (31) 1 mo.	98/37 (57)	26/5 (12)	24/8 (13)	21	Excellent
14. T. S. 11/7/49	43/3 (16)	42/12 (22)			27/5 (12)	-	23	Excellent

data have gradually been obtained in fourteen patients ranging from five to twenty-three months after surgery (Table IV). It is well known that readjustment of the pulmonary vascular circulation, following valvular surgery, may take considerable time, hence the changes noted within the first postoperative month do not necessarily reflect the ultimate pressure or, for that matter, clinical level. Case 11 exemplifies this point. Despite marked improvement in the patient's clinical subjective status at seven months the pressures were significantly higher than those obtained preoperatively. At sixteen months they returned to slightly under the original level representing no essential objective improvement in direct contrast to her very excellent clinical state which permits her to live a normal life, working full time.

Perusal of Table IV will reveal that eleven patients (all but Cases 1, 3, and 11) obtained very satisfactory and significant lowering of the pulmonary and right heart pressures. Surprisingly, in eight of these, the drop was practically to normal levels, representing in several a decrease of 50 to 80 mm. Hg systolic. In each of these eleven the subjective result followed suit so that all are now classified as excellent results by the criteria set forth in the text. One of the remaining three (Case 1) is unimproved clinically after temporary initial relief. Another (Case 3) is improved clinically to a much greater degree than his pressure changes would indicate. The third (Case 11) obtained an excellent result as discussed previously.

The electrocardiogram frequently aids in determining the candidacy of these patients, especially when there is more than one valve defect. Evidence of right ventricular enlargement suggests that the predominant lesion is one of mitral stenosis. It is to be noted that a right axis deviation (plus 90 degrees or more) was present in 140 cases, a normal axis in 23, but more particularly, that in only one instance was there evidence of a left axis shift. In this case, a stage V, mitral insufficiency was found to be equally as prominent as stenosis when the valve was explored. Commissurotomy, although accomplished adequately, had no effect on the course of the irreversible cardiovalvular and pulmonary vascular disease and she continued unimproved until death, ten months later. Finding a left axis shift predicates that other factors are at play and a careful reevaluation must follow.

Operative Findings.—Seldom in the past has there been the opportunity to examine at firsthand the nature of living valvular pathology in situ. It has been a source of wonder to us to find the average stenotic valve in relatively good condition in contrast to the incapacitation of the patient (Table V).

1. The size of the valve orifice, as estimated by the tip of the exploring index finger in 133 of the cases, was approximately 0.5 to 1.0 cm. in diameter or, in more readily appreciated terms, the size of one to two heads of kitchen matches. The cusp margins were thickened and fibrotic with fixation of the periorificial ring. Nevertheless, just enough pliability remained in the valve leaflets so that in only twenty-nine cases could a regurgitant jet of blood be detected with ventricular systole. In the remaining twenty-nine (two were not

TABLE V. OPERATIVE FINDINGS

Size of valve orifice (finger estimation)	
(a) 0.5-1.0 cm. (minimal regurgitant jet in 29)	133
(b) 1.1-1.5 cm. (no jet in 6)	16
(c) 1.6-2.0 cm.	8
(d) 2.5-3.5 cm.	5
	162*
Orifice after commissurotomy	
(a) 4.5 cm.	3
(b) 3.0-4.0 cm.	131
(c) 2.5 cm.	23
(d) 1.5-2.0 cm.	5
	162*
Calcification of valve	75

*Two valves, size not recorded.

recorded) the orifice was somewhat larger. The diameter of sixteen was 1.1 to 1.5 cm., and in six of these no insufficiency could be appreciated. Eight fell into the 1.6 to 2.0 cm. size, three at 2.5 cm., and two were between 3.0 to 3.5 cm. Some of these were obviously poorly selected cases as the result obtained hardly justified surgical intervention.

In contrast to the firm and indurated cusp margins, the valve leaflets, although thickened, were considerably more flexible. Their consistency was usually that of kid glove skin, less frequently like shoe leather. Only twenty had lost all semblance of a functioning valve in that they were completely fixed and rigid. Extreme fibrosis and calcium replacement were responsible for this effect. Thus, as a rule, the "mitral megaphone" could be felt to ride back and forth as a complete cone in response to changes in pressure on its two sides, always maintaining its obstructing effect and unable to open or close at its orifice.

It was possible, despite the pathologic nature of the valve, to accomplish a satisfactory separation of the valve leaflets in 157 cases. Three of these were opened to 4.5 cm. in diameter, 131 between 3.0 and 4.0 cm. (optimum), and 23 others to 2.5 cm. In five the conditions present would permit enlargement to only 1.5 to 2.0 cm. Ideally, the objective of commissurotomy is not only to enlarge the stenotic orifice, but also to restore a considerable measure of valve function. This latter goal could not be attained in approximately twenty-five cases because the valve tissues were replaced by fibrotic scar and calcium to such a degree that all pliability, even after release of the cusps, was lost.

The technique of commissural separation, as used by us, has been repeatedly described in previous writings. Both the right index finger and the specially devised guillotine knife are as a rule introduced simultaneously through an opening near the tip of the purse-stringed left auricular appendage. Whenever possible, after palpation of the entire valve for orientation, one small cut is made across the rind of the anterolateral commissure after which gentle finger pressure is applied. This will usually accomplish dissolution of the commissure to the myocardial wall. At any point where undue

finger pressure is necessary to divide the adherent leaflets, an additional small cut is made facilitating rapid and clean division without the danger of tearing or avulsing either the valve leaflets or their attached chordae tendineae. An anterolateral commissurotomy of this type is, in the majority of instances, sufficient to accomplish maximum results both clinically and physiologically. In a very considerable number of cases one small additional cut was made into the fused and rolled posteromedial commissure just to transect the rigid ring. This was not carried widely into pliable valve tissue, for the danger of producing major regurgitation in this area is great. In the past six months the posteromedial commissure has rarely been opened unless adequate anterolateral separation has been impossible. The over-all result would seem to be better by observing this precaution. When one has become completely familiar with the proper use of the guillotine it will become obvious that its use is essential in perhaps 30 per cent of cases. In an additional 20 to 40 per cent a more complete and thorough commissurotomy can be performed (assuring the patients of a more certain, long-term future) when this technique has been mastered. On the other hand, when thrombotic material is present in the appendage it has been our practice to insert the finger alone, hoping that definitive surgery of an adequate nature can be done by this method in order to obviate the extra manipulation of both knife and finger, lest an embolus from the appendage be produced. Very often, however, the valve has required additional incision and without removing the finger the guillotine is slipped in beside it.

The combination of finger and knife, in the actual division of the commissures, has been used in 114 cases, the knife only in 21, and the finger alone in 28 cases. One was not recorded (Table VI). The anterolateral commissure was divided alone in eighty instances, both the anterolateral and posteromedial in seventy-three, and the posteromedial by itself in four. In six cases only dilatation without separation could be accomplished, in five with the finger and in one with a Kelly hemostat (tiny appendage in a 4-year-old child). One case was not recorded adequately.

2. Calcification was present in some measure in seventy-five of the valves (Table V). The frequent site for such infiltration was on the anterior valve

TABLE VI. TECHNIQUE OF COMMISSUROTOMY—164 CASES

Method		
Finger and knife		114
Knife only		21
Finger only		28
(a) Finger dilatation only	5	
(b) Kelly dilatation only	1	6
		163*
Commisure		
Anterolateral		80
Anterolateral and posteromedial		73
Posteromedial		4
Dilatation		6
		163*

*Technique not recorded, one case.

leaflet over its medial aspect—that portion nearest the outflow tract of the left ventricle. Nineteen of the valves were lightly flecked with tiny calcific beads along the surface of the cusp margins. In forty-four the calcium extended back into the leaflets for varying distances at one or more points of the circumference. Even in these situations considerable mobility could frequently be restored by commissurotomy. In twelve cases calcium was present in larger quantity, practically encircling the mitral orifice and appreciably limiting the reconstructive effect obtainable by incising the commissures. Surprisingly, although little if any valve function could be restored in this group, generous enlargement of the orifice straight through the calcific crust produced an excellent result in four, with considerable improvement in four others. The remaining four were unimproved. Fortunately the anterolateral commissure is less frequently involved by calcium, and separation at this point will improve the patient, as a rule, regardless of the status of the remaining valve cone. It is to be emphasized, therefore, that the presence of calcium per se need not militate against a good functional result. Occasionally, without detectable calcium, the circumferential contraction of the atrioventricular ring, together with extreme thickening of the valve cone and foreshortening of the chordae tendineae, result in such fixation that commissurotomy is to no avail.

3. Gross or microscopic evidence of thrombosis was encountered in the left auricular appendage of thirty-seven patients. Preoperative arterial embolization was associated in only ten of these, and thirty-three were in auricular fibrillation. There was one questionable embolus produced at the time of surgery despite the fact that the finger was introduced through the appendage after proper preparation in each of the thirty-seven cases (see discussion which follows).

Immediate Postoperative Status (Table VII).—

Chest pain: (a) The usual thoracotomy pain, intercostal and chest wall in type, is to be expected and was experienced by all patients.

(b) In addition, pleuropericardial pain may be a striking sequel to this surgery and may be much more severe and persistent than that experienced by the average thoracic patient. It occurred in 38 per cent of cases and may be most prominent during the second postoperative week. Usually it recurs sporadically during the ensuing six months. The pain is typical of that seen in acute pericarditis and similarly is aggravated by swallowing, deep inspira-

TABLE VII. POSTOPERATIVE STATUS—164 CASES

Pleuropericardial pain		63 (38%)
Pericardial effusion		5
Sinus rhythm to auricular fibrillation		23
(a) Transient	8	
(b) Permanent	15	
Auricular fibrillation to sinus rhythm		6
(a) Transient	1	
(b) Permanent	5	
Postoperative hemiplegia		2
Reactivation of rheumatic infection (??)		0

tion, and change of position. The location of the pain may be in the lower substernal area, either localized or radiating to the neck or back. At times a small pleural effusion or early signs of pulmonary congestion may develop.

The importance of this pain lies in its characteristics of persistence, severity, unpredictability, and its partial response to salicylates but not to antibiotics, and ordinary doses of narcotics. In several extreme instances this distress persisted daily for three months, precipitating right heart failure. Such a morbidity is rare and, fortunately, in these instances recovery has been complete and the patients have returned to normal activity. An attempt has been made to anticipate the probable onset of this complication by investigating the patient's rheumatic state prior to surgery. It has been impossible to predict with any certainty such complications, however, either from the history, the physical examination, or laboratory studies.

Pericardial effusion: There were nine instances in which pericardial aspiration seemed desirable. No fluid was obtained in four, less than 100 c.c. in four, and 150 c.c. in one. This type of effusion is not associated with the usual signs and symptoms of cardiac tamponade. The exhibition of undue anxiety associated with a feeling of strangling and oppression should arouse suspicion. The apical rate may be normal or slightly elevated; moderate orthopnea and hyperpnea may develop with profuse perspiration, and as a rule the systolic blood pressure is lowered. The jugular veins infrequently are distended. Even as the fluid is withdrawn the patient may experience subjective relief. Repeated aspiration has not been necessary.

Arrhythmias: Auricular fibrillation developed postoperatively within the first ten days in twenty-three individuals who had had sinus rhythm prior to surgery. Eight were transient and returned to a sinus mechanism with or without quinidine, and fifteen became permanent.

There were six instances in which auricular fibrillation reverted to sinus rhythm after surgery. Only one of these has returned to a state of fibrillation.

Postoperative emboli: Two patients awoke from anesthesia with a hemiplegia. No evidence of gross or microscopic thrombosis was present in the left auricular appendage in either so that the origin of the embolus was unknown, possibly from the valve itself. One-third, in whom the appendage was obliterated by thrombotic material, developed pain in the popliteal area of the left leg shortly after surgery. This patient had had innumerable emboli to the brain, viscera, and extremities in the past, had undergone an attempted embolectomy in the right femoral artery several years before, and had been on Dicumarol for many months prior to admission. Bilateral intermittent claudication had been present for a long time. A therapeutic caudal block was continued for two days without noticeable effect as the left leg became more and more discolored with signs of demarcation below the knee. The right foot was only slightly cyanotic, nonpainful, and maintained unlimited function. Left femoral artery exploration revealed a good but not pulsatile flow of blood and no embolus could be located either above or below from the aortic bifurcation to the pop-

liteal artery. Eventually, a mid-thigh amputation was necessary and the patient made a fine recovery. Extensive search of the vascular tree in the amputated limb failed to discover fresh embolic material, although all vessels were thickened and contracted as in thromboangiitis obliterans.

Reactivation of rheumatic infection: In our considered opinion there has been no instance of an exacerbation of acute rheumatic fever as ordinarily seen in children. This was somewhat unexpected in view of the histologic evidence of the rheumatic state in 49 of the 143 appendages examined microscopically. No doubt a considerably higher incidence of rheumatic stigmas would have been found had the myocardium proper also been examined. This opinion is felt to be true in view of the lack of evidence of carditis exemplified by the development of additional valvular lesions. No change has occurred in the valvular status other than that which was produced traumatically, namely, a mitral systolic blow. In addition, using the major and minor criteria of Jones for the diagnosis of rheumatic fever and its activity, migratory polyarthritis, fever responding to salicylates, subcutaneous nodules, routine increase in the sedimentation rate, and the like have not developed. One outstanding complication is at least under suspicion as being of rheumatic etiology, namely, pleuropericarditis, which has been temporarily present in varying degree during the first six postoperative months in sixty-three patients. Four patients had a continuation of fleeting and migratory bone and muscle aching present for years but this was not made worse or actually affected one way or the other by surgical intervention. The two children in the series did fulfill the generally accepted criteria for rheumatic activity but in all probability were in an active state at the time of surgery. Further observation will be necessary before the role of rheumatic infection as influenced by surgery can be evaluated.

Auscultatory changes: A loud pericardial friction rub was almost invariably heard immediately after surgery, disappearing slowly within ten days.

The mitral diastolic rumble becomes greatly diminished in intensity, and during the first seven to ten days may be inaudible.

In most of the cases a mitral systolic murmur was produced. Its usual intensity may be graded as 1 to 2 and this murmur usually persists. It is felt that as a rule this murmur does not represent a significant degree of insufficiency.

The sharp and accentuated mitral first sound usually remains unchanged in quality, even though the diastolic rumble may disappear. This sound appears to be the least affected by mitral valve surgery. The pulmonic second sound frequently remains accentuated, even though the pressure in the pulmonary artery has been reduced.

Thus, to summarize, the murmurs may change materially but the individual heart sounds may not.

Present Functional Status.—There is no more difficult task in the compilation of clinical data than to reach agreement on suitable and self-explanatory criteria for the evaluation of surgical results. This is especially true when the

pathologic and physiologic pattern of a disease entity, such as mitral stenosis, is so varied. The common ground for comparison would seem to be a functional status of the patient observed at least six months or more after surgery.

Table VIII summarizes the results as obtained in this series. Eighty patients are classified as being excellent in that they have been restored to a normal, productive life, enjoying normal activities without obvious limitation. Limitation of diet and mercurial diuretics are no longer necessary. Fifty-four are still taking digitalis, in most instances, however, because neither physician nor patient has gotten around to discontinuing it. Forty-five are considered to be objectively and subjectively improved, as evidenced by their return to almost normal life and activity within their own particular limitations, determined on a trial and error basis. Most of this group are under medication as a precaution because of the demands of their more greatly enlarged sphere of activity. A few, terminally incapacitated, have now become so amenable to therapy that reasonable activity requires digitalization only. The range of improvement in this group varies widely with the condition of the valve found at surgery and the degree to which valve function could be restored. Their progressive downhill course has been abruptly terminated or reversed, some to regain a high level of efficiency, and others to remain on an improved plateau.

TABLE VIII. PRESENT FUNCTIONAL STATUS*—164 CASES

STATUS	NUMBER	PER CENT	
Excellent	80	48.7	76%
Improved	45	27.4	
Unimproved	20	12.1	
Deaths			
(a) Operative	10	6.0	
(b) Late (8, stage V) (1, stage III)	9	5.4	

Nineteen patients have been essentially unimproved. Seven of these had multivalvular disease with significant mitral insufficiency, the seriousness of which was appreciated only at the time of valve exploration. Psychoneurosis of major degree became more prominent after surgery in four, making cardiac evaluation difficult, although three were considered improved from a circulatory standpoint by their referring physicians. In three there was aneurysmal dilatation of the left atrium, with associated mitral insufficiency. The pathologic nature of three other valves was such that a satisfactory commissurotomy could not be accomplished. The inadvertent production of significant amounts of insufficiency by opening the posteromedial commissure too widely militated against a good result in the final two.

The operative mortality (Table IX) in the 164 cases under complete analysis was 6.0 per cent (ten cases). As might be expected, the deaths all occurred in the more advanced stages of the disease (stages III, IV, and V). In the 105

TABLE IX. CAUSES OF DEATH—164 CASES

OPERATIVE			LATE	
Technical		5	Irreversible cardiovalvular and pulmonary vascular disease with progression into terminal failure.	
(a) Split cusps (finger)	2		(all in stage V)	8
(b) Medial commissure			Pre-existing insufficiency increased by surgery	1
(1) Cut too far	2			
(2) Unexplained	1			
Patients near extremis		2		
Cerebral anoxia during surgery		2		
Unsuspected associated aortic stenosis (forward failure)		1		
Totals		10		9

cases in stage III, five deaths occurred (4.7 per cent). For the most part these were due to faulty technique early in this series when experience was minimal. Two were the direct result of the finger pressure method which accounted for the inadvertent splitting of a valve leaflet (one) or partial avulsion of one leaflet from the myocardial wall beyond the commissure (one). In one other there was no anterolateral commissure present and wide incisional separation of the posteromedial commissure produced a lethal insufficiency with the patient dying three weeks after surgery. Hypotension throughout surgery with its attendant cerebral anoxia was responsible for death within two hours of an otherwise satisfactory technical commissurotomy. The fifth death was due to forward failure in a patient who at autopsy proved to have an unsuspected severe aortic stenosis in addition to the mitral stenosis for which she was operated upon.

The cause of three deaths in the stage IV group was not entirely clear. It was thought that one was due again to hypotension and cerebral anoxia as the patient died without reacting in six hours. Post-mortem examination revealed a generalized cerebral edema and nothing else to account for death. The second case was totally unexplainable in a patient who should have done well and in whom autopsy was not permitted. The third was brought about by the surgical production of too much regurgitation from a bilateral commissurotomy in which the posteromedial commissure was opened too widely. It was further complicated by a left hemiplegia.

Both fatalities in the stage V cases were in patients in the extreme terminal state. One was in uncontrolled congestive failure and was done under great pressure. The other required continuous oxygen in the semi-Fowler position, and yet seemed free of clinical decompensation. Examination later uncovered their extensive pulmonary fibrosis and generalized irreversible cardiorenal-hepatic changes.

There were nine additional deaths which occurred from one month to two years after surgery (Table IX). All but one of these were grouped as terminally incapacitated, irretrievable cases by the classification outlined previously. One patient died from multivalvular disease with mitral insufficiency increased by surgery. The other eight, despite an adequately performed valvular operation in keeping with the pathologic conditions encountered, died because irreversible cardiovalvular and pulmonary, hepatic, or renal vascular changes were present, which no mere relief of mechanical obstruction could alter. They rep-

resent failures on the part of an unsuspecting medical profession. At this stage in the development of cardiovalvular surgery they are entirely excusable and may in a sense be regarded as tragic examples with which we all must be re-educated. With a greater understanding of the inherent nature of progressive mitral stenosis and the clear demonstration of a technically successful operation, both as to a low mortality and the satisfactory results obtainable, the selection of patients at an early stage now becomes the paramount duty of every practicing physician and procrastination can no longer be tolerated.

Indications for Commissurotomy.—In the light of the foregoing presentation, the selection of patients for surgical intervention can be simply stated. The ideal candidate is the patient with pure mitral stenosis and beginning symptoms of cardiopulmonary dysfunction such as shortness of breath upon exertion. Fatigue out of all proportion to the patient's physical activity is frequently a prodromal or accompanying finding. At the moment the mere presence of a well-defined mitral diastolic murmur without accompanying symptoms is regarded by many as insufficient reason to suggest surgery. No doubt the day will come in the very near future (if not at present) when such a course will seem unreasonable. No one today awaits the complications and end results of mechanical deformities such as patent ductus arteriosus or coarctation of the aorta before proceeding to their surgical correction. On the contrary, it is the accepted operative practice to proceed shortly after the diagnosis has been made clear and long before symptoms and signs of irreversible damage have supervened. Surgery for mitral stenosis at such an early stage would carry an infinitely lower mortality than it does today and much cardiac disability might be obviated.[18, 19]

All other indications are merely compromises from the ideal, but for years to come an understanding of the more advanced but altogether salvable states is essential. An outline under seven major categories (and contraindications) will most succinctly serve this purpose:

1. History
   (a) Early cardiopulmonary dysfunction—ideal
   (b) Marked dyspnea, hemoptysis, reversible failure—acceptable
2. Age—elastic range, physiological rather than chronological
3. Valvular defect
   (a) Pure mitral stenosis—ideal
   (b) Associated mitral insufficiency and/or aortic valve lesion in presence of normal left ventricle—acceptable
4. Roentgenogram
   (a) Left atrium and right ventricle minimal enlargement—ideal
   (b) Minimal left ventricle enlargement—questionable but acceptable
5. Electrocardiogram
   (a) Normal electrical axis or right ventricular strain
   (b) *Never* left axis shift
   (c) Auricular fibrillation with controllable ventricular response—acceptable
6. Functional capacity
   (a) Stages I and II, statically incapacitating at the most—ideal
   (b) Stage III, progressively incapacitating—acceptable
   (c) Stages IV and V, debatable—occasional good result

7. Complicating factors
   - (a) Arterial embolic episodes
   - (b) Recurrent hemoptysis
   - (c) Hypertension (?)

   } Acceptable, and may be urgent

8. Contraindications
   - (a) Acute rheumatic fever
   - (b) Subacute bacterial endocarditis until controlled
   - (c) Associated marked mitral insufficiency or aortic lesions with all cardiac chambers enlarged

In conclusion, surgical reconstruction of the stenotic mitral valve is a major adjunct and an absolute essential in the continuing care of these rheumatic victims. Although such patients can and have been restored to a high level of efficiency and, as such, enjoy a more normal life, nevertheless, if referred for surgery late in the course of their disease, they must always be considered as cardiac patients under the watchful care of their attending physicians just as the surgically treated tuberculous patient will require frequent observation, advice, and management. The burden of selecting patients at an early stage rests squarely on the shoulders of both physician and surgeon and this responsibility can no longer be avoided or postponed.

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DISCUSSION

DR. CONRAD LAM, Detroit.—I wish to congratulate Dr. Glover and his group on continuing the work begun with Dr. Charles Bailey. I should like to discuss his paper by presenting a very small contribution to technique which we have found helpful and which will, I believe, affect the results.

(Slide.) This is what we call a six-fingered commissurotomy glove. Those who have done commissurotomies by the technique of the authors of the preceding paper realize that there is sometimes considerable hemorrhage. Blood flows between the two gloves along the knife and escapes through the slit in the outer glove. At the suggestion of Dr. Edward Munnell, we have added to the ordinary glove a sixth finger on the palmar surface through which the knife can be passed and around which a tight ligature can be placed. Then the surgeon can take his time, deciding whether or not to make a cut here or there. Actually, the knife can be rotated until its flat surface is perpendicular to the finger and then the cutting edge can be palpated. This is particularly helpful in incising the medial commissure, when orientation is especially important.

(Slide.) This photograph taken during a commissurotomy shows the finger in the opening in the auricle, hemostasis being maintained by the snare. This is the palm of the glove which is filled with blood, and not a drop escapes.

(Slide.) The construction of the glove is simple. Details will be reported by Dr. Munnell in an early issue of the JOURNAL OF THORACIC SURGERY.*

I should like to mention one embolic episode in an otherwise uneventful commissurotomy. When the patient was being transferred to the stretcher, it was noted that both legs were cadaveric in appearance. The patient was lifted back onto the table, the instruments were quickly sterilized and, through a retroperitoneal approach, a saddle embolus the size of a small egg was removed. The patient now has normal circulation in the legs.

DR. CHARLES P. BAILEY, Philadelphia.—I should like to present a common complication in surgery for mitral stenosis.

(Slide.) This is a picture of the brain destruction following arterial embolization. This nearly always involves the middle cerebral artery when it occurs during commissurotomy, or I guess at any other time. You can see why these patients have residual paralysis of one side if they survive—why they have a degree of aphasia if this damage affects the left side of the brain.

(Slide.) This is a diagram representing the insertion of a finger through a clotted appendage, dislodging particles which can cause peripheral arterial embolization. Approximately 30 per cent of the patients who come to commissurotomy have some degree of thrombosis in the appendage, so that this accident is not completely preventable in all cases.

(Slide.) This is the other major source of arterial embolization—calcium deposits on the edge of the valve which may be inadvertantly dislodged at the time of cutting, or of breaking the commissures with the finger. Often this calcium is like loosely attached sand; it rolls under your finger and you know you have released small particles into the circulation.

Entrance into the heart by way of the left superior pulmonary vein can be made. I have made them in about twenty-three instances. However, two patients died as a result of that technique and so it seems to be more dangerous than embolization to the brain itself. In a personal series which I worked out last November there were twelve cases of cerebral

*Munnell, Edward R.: Six Fingered Glove for Mitral Commissurotomy, J. THORACIC SURG. 23: 628, 1952.

embolization in 235 operations, an incidence of about 5 per cent. Eight of those patients died, mainly as a result of that embolization. Since November, 1951, a series of 157 patients have been operated upon by a technique with modifications as shown.

(Slide.) This represents the circulation to the brain and you can see that the middle cerebral arteries, which are the site of most of the emboli, are direct continuations of the internal carotid arteries. Obviously, if you could obstruct them during the course of the intracardiac surgery you would not have any trouble with cerebral embolization. In my entire experience with approximately 500 cases I have had only one case of embolization to the lower extremities during commissurotomy, so it is obvious that the little emboli that undoubtedly get to the vessels of the lower extremities do not necessarily cause any trouble. The main problem is to protect the brain.

(Slide.) This is what we have worked out. It may not be the best way, but it is the way we did it. We mobilized the left common carotid artery in the mediastinum above the aortic arch, and placed a piece of umbilical tape around it. Then at any time in the surgery the umbilical tape can be pulled up, kinking the vessel so that no blood flow occurs. We think that this can be done for at least three minutes, while the right innominate artery is completely blocked, without causing any cerebral damage. Since the mediastinal portion of the innominate artery does not lend itself well to kinking, we had to adopt a more definitive technique of management. A bulldog clamp is placed across the origin of the innominate artery at the beginning of intracardiac surgery and is left on throughout that portion of the surgery, in one case, 34 minutes. I should like to report that we have had no complications while using this technique, but that is not quite true. In spite of using this technique in 157 consecutive patients, we did have one case of left-sided cerebral embolization. I suppose a clot may have lodged there either between our periods of traction upon the umbilical tape, or perhaps after we were through. We thought we were adequately protecting the brain by traction upon the umbilical tape whenever we were doing anything inside the heart and for fifteen heartbeats thereafter, but this patient still developed a left-sided embolus.

We also had one other case with a cerebral complication, although I do not believe it was due to an embolus. This man previously had had a right-sided cerebral embolus and he had a left auricle almost completely full of clot. We were so afraid of a repetition of the embolus that we made longer traction on the left common carotid perhaps than we should have, about five minutes. With the previously diminished right cerebral circulation, and the innominate being clamped off, he has developed an exaggeration of the previous paralysis on the left side. Therefore I think we may have to limit our time of carotid obstruction more strictly.

DR. HENRY SWAN, Denver.—I cannot resist paying tribute to the wonderful work which Dr. Glover and Dr. Bailey have done in pioneering this operation for mitral stenosis. I should like to make one brief comment concerning the selection of patients. Dr. Glover has ably demonstrated that operation can be done at low risk and certainly should be done early in the course of this disease. It is generally agreed, I believe, among pathologists, that in the presence of long-standing venous and associated arterial pulmonary obstruction, changes occur in the pulmonary vascular bed which eventually become irreversible and which maintain, of themselves, the pulmonary hypertension. In the course of mitral stenosis, therefore, there comes a time when operation can be done only at a much increased risk and without promise of much benefit to the patient. My colleague, Dr. Blount, and I have been interested in studying this phase of the problem, and have been doing so by attempting to evaluate the response of the pulmonary circulation to exercise. In the normal individual who exercises, of course, the pulmonary artery pressure does not rise materially and the cardiac index goes up sharply. In the patient with mitral stenosis the pulmonary artery pressure, which may be slightly or moderately elevated at rest, on exercise shows a very pronounced rise and the cardiac output does not show a very great increment. This rise in pulmonary arterial pressure would appear to be the result of two components. The first is the obstruction at the venous end, that is to say, the obstruction of the valve itself; this can be measured by the use of the so-called "pulmonary capillary pressure." The other component

is the resistance of the pulmonary vascular bed itself; this can be measured by subtracting the pulmonary capillary pressure from the pulmonary artery pressure. It appears likely that in most patients who have a pulmonary artery pressure rise due primarily to and associated with a marked rise in pulmonary capillary pressure, the valve itself is causing the pulmonary artery rise and operation would be of benefit. On the other hand, if a marked pulmonary artery pressure occurs, a rise in pressure which is not associated with a pulmonary capillary pressure rise, then it seems reasonable to assume that the peripheral resistance in the vascular bed of the lung itself is causing this rise, and that operation on the valve would would not be of great benefit to this patient.

We have doing lung biopsies at the time of operation in the attempt to correlate these physiologic observations with the observed changes in the pulmonary arterial tree. It is still too early for us to be able to say whether this concept has validity, but I believe that if so, it may be of some help in the selection of patients and in the prognosis of what benefit may be derived from the operation.

DR. HARRISON BLACK, Boston.—I should like to congratulate Dr. Glover on his excellent series and reported gross mortality of 7 per cent. However, we on Dr. Dwight E. Harken's services at the Peter Bent Brigham, Boston City, and Mt. Auburn Hospitals are impressed with the advisability of a breakdown of mortality statistics. Dr. Glover's results are certainly commendable in the aggregate. However, to tell certain patients that they have seven chances in 100 of dying as a result of the operation would be excessively pessimistic. On the other hand, some patients have nowhere near 93 chances in 100 of surviving. Mortality statistics should serve to tell us how well or how badly we are doing in any particular group of patients so that we may make intelligent decisions in the choice of patients for operation. Patients who have mitral stenosis range the spectrum of illness from no disability in athletes to those who succumb to their disease. The results we get in survival and rehabilitation depend on the part of the spectrum in which we are working, what we do in this area, and how we take care of the patients before and after operation.

Obviously, the starting point for such an analysis is a classification. (Slide.) All patients with mitral stenosis that we see are placed in one of four clinical groups. This is done preoperatively without regard to factors that might increase the operative risk, such as calcified auricular thrombus, previous history of peripheral or pulmonary emboli, calcification of the mitral valve, or the existence of other valvular lesions.

Group I is the one we consider *benign*. In it are placed all patients who have the murmur of mitral stenosis but who are not disabled by it. Because many of these people have a good prognosis and may lead an active and useful life we do not advise them to have an operation. They must, however, be followed carefully lest they slip into a more advanced group.

Group II consists of those who have various degrees of incapacity, predominantly due to dyspnea, but who are getting no worse. In this *disabled* group we have only recently accepted patients for operation, for we previously felt that they were functioning adequately enough to wait until our technique had become stable. It has now done so. To some of the patients in this group whose incapacity interferes with their ability to earn a living or to live a life acceptable to them, we are now offering mitral valvuloplasty.

It is in Group III, the one we call *hazardous* because of the poor prognosis on medical management, that we feel the operation has the most to offer in terms of rehabilitation at an operative risk that now compares favorably with that of any major abdominal procedure. This is the group whose dyspnea and limitation are progressing. Many of them have had repeated episodes of pulmonary edema brought on by the slightest exertion or excitement; many have had hemoptyses or emboli; a large percentage of them have fibrillation; and they are generally on digitalis, limited salt intake, and mercurial diuretics. In spite of the best of medical management they are becoming more disabled. It is important to have this clinical picture in mind when we consider the mortality figures, for these patients are well down on the scale of severity of disease.

It happens that we have operated on a large number of patients in Group IV. This is the group of *terminal* patients who are rapidly failing, frequently are in irreversible failure, and have considerable damage to myocardium, lungs, and liver. Many of them are able to exist only at a bed-and-chair level of activity.

(Slide.) In this slide is illustrated the breakdown of mortality according to the groups as outlined. As you can see, there have been 257 total operations for mitral stenosis in this clinic. Many of the earlier procedures have now been abandoned. Eliminating these there have been 242 finger fracture valvuloplasties. In Group I there have been no operations. In Group II, the handicapped but not progressive cases, there have been 14 operations with no deaths. In Group III, the hazardous group, we have done 130 valvuloplasties up to the present time with six operative deaths and no late deaths, giving a mortality, operative and late, of 4.6 per cent. In the last 100 patients in this group there have been only two deaths, one in a patient with a solidly calcified auricular clot and another apparently from adrenal exhaustion.

In considering the terminal Group IV patients one can appreciate how desperately ill they are from the medical mortality. Early in our experience we had the opportunity to observe 19 Group IV patients who were classified by exactly the same criteria as the others but who refused operation. Seventeen of these died within a year, 15 within six months, giving a medical mortality of 89 per cent. Another fact worth mentioning is that in a few instances we have felt that a patient was too ill to accept for operation. None of these has survived for more than a week after this decision. In Group IV patients we have done 98 valvuloplasties with an over-all mortality, operative and late, of 31 per cent. Expressed another way, we have been able to salvage 69 per cent. In the last 45 procedures in this group the mortality has been reduced to 20 per cent.

The point of all this is to demonstrate the marked contrast in risk and salvageability in the various stages of the disease, mitral stenosis. Obviously it is not best to operate in the terminal group; we do not like to do so. We are, however, forced to it quite often because some internists wait until the patient gets into this desperate condition. It is imperative that we educate our medical colleagues to refer these patients for operation at a time when there is a reasonable chance to do something for them. It can no longer be said that the operative risk justifies a policy of watch and wait when the patient is obviously getting progressively worse.

Just a word about the technical problems of management that face us at present. We can agree with Dr. Bailey that the *bête noire* of mitral stenosis surgery now is the production of emboli either of blood clot or calcium particles from the valve at the time of the intracardiac maneuver. We have not obstructed the carotid arteries in the chest but have had the anesthetist obstruct them in the neck. I do not have the figures, but believe that the embolization problem has been somewhat improved by that method. We have also allowed a brisk flushing of blood out of the auricle before insterting the finger as suggested to us by Dr. John Jones, and in several instances have washed out large clots in this way.

In the Group IV patients the problems are no longer associated with the operative technique. We have reached the era, as I believe most everybody has, in which we can operate with safety and few technical errors. The problems are those of management of the pre- and postoperative fluid and electrolyte balance, management of arrhythmias, and the conduct of the anesthesia. Many of these patients have severe sodium depletion because of long periods of restricted sodium intake and mercurial diuresis on frequent occasions, and some of the Group IV patients have actually died of intractable electrolyte imbalance while attempts were being made to prepare them for operation.

In the field of anesthesia we feel that the efforts of Dr. William Derrick in keeping the patients adequately ventilated with his respiratory assister, and also in using a minimal amount of anesthetic agent, have unquestionably allowed us to operate successfully on patients who would have died with any other anesthetic techniques.

In the postoperative period the Group IV patients often present a severe form of electrolyte imbalance which is a marked exaggeration of the normal response to operative trauma. We have frequently found that the serum sodium level falls to 120 to 125 milliequivalents per liter, and on some occasions we have found values as low as 108. Not all these patients show clinical evidence of the low sodium syndrome, such as persistent hypotension, weakness, and apathy, and complete balance studies in one patient have shown that these low serum figures are not necessarily a true reflection of the total body sodium. However, when the clinical syndrome is associated with a low serum sodium value, we have restored these patients to compensation by the administration of hypertonic saline. We have been immeasurably aided in our management of this problem by Dr. Francis D. Moore and his laboratory associates. On other occasions evidence of adrenal exhaustion has been present and corrected with ACTH or cortisone.

One further point requires comment. Dr. Glover has said that 75 per cent of the patients in his series are improved postoperatively. Our experience has been somewhat different, for in our series no surviving patient has failed to be improved, some of them to a miraculous degree. We feel that when mitral stenosis is severe and is adequately corrected the patient will manifest at least some improvement despite damage to liver, lungs, and myocardium if he survives. Admittedly this improvement may be less than if he were operated upon before "the horse is stolen." We take no personal credit for this; it is probably a matter of diagnostic precision in the selection of patients. For this excellent cardiac collaboration we express our appreciation to Drs. Laurence B. Ellis, Lewis Dexter, and Samuel A. Levine.

DR. DENTON A. COOLEY, Houston.—In our experience at Baylor University with thirty commissurotomies we have had one death. I would like to cite one case which illustrates the application of commissurotomy during pregnancy. This patient was 28 years old and in the fourth month of her first pregnancy. During the past four years she had developed progressive signs of incapacity and had several bouts of pulmonary congestion consequent to mitral stenosis. Commissurotomy was performed on Nov. 2, 1951, and she recovered uneventfully. She is expected to deliver in the next few days, and I understand she has remained active throughout pregnancy. (On May 18, 1952, after a six-hour labor she delivered a 7 pound 3 ounce female infant, and the postpartum course was uneventful.)

At the time of operation I knew of no other such experience, but Mr. Laurence Whytehead reports to me that Mr. Brock of London has treated one case similarly, and I understand Dr. Rumel of Salt Lake City has another. If the combination of pregnancy and mitral stenosis becomes an indication for commissurotomy, I suggest that it be done before the fifth or sixth month, after which the demand upon the left ventricle increases sharply with increase of the placental circulation.

DR. F. E. TRICERRI, Buenos Aires, Argentina.—I should like to report my experience in Argentina in twenty cases of mitral stenosis. Of those, fifteen belonged to Group III, of Harken classification, and there were no deaths in those. In five cases that belonged to Group IV, there were three deaths, due to embolism in the cerebrum. I would like to ask Dr. Glover how to avoid cerebral emoblism. Of those three patients one had five previous embolisms, one had two, and the third had one. In all three cases the appendage was full of blood clots and to wash it out I let blood through the appendage before introducing my finger; I was unsuccessful in coping with the emboli which finally caused death, one in seventy-two hours, one forty-eight hours, and the other within ten days.

DR. GLOVER, (Closing).—I am very grateful to the discussers for their comments. I should like to refer to each in turn if time permitted, but I have been asked to show a strip of film depicting the intracardiac portion of a mitral commissurotomy which would perhaps be of more interest. In passing, let me take exception to the use of the word "benign" in Dr. Black's classification. The word is misleading for in most instances the condition will not remain benign for long and therefore tends to convey a wrong impression.

(Movie.) Many people have asked, "What goes on inside the heart?" They all know how to get to the appendage, so we have prepared this heart specimen of a 24-year-old girl

who came into the hospital during her first attack of pulmonary edema and died within twenty-four hours before surgery could be undertaken. This is the size of the stenotic valve orifice most frequently seen. It is tiny, as you see, and will just admit the head of a paper match. The patient was in extremis, of course, when first seen, and we show this primarily to illustrate that even in a terminal state the valve tissues are often quite pliable. This is the anterior or aortic cusp and here you see the posterior leaflet. They are not the thin cellophane-like normal structures but thickened like the skin of a kid glove. There is plenty of pliability provided an adequate commissurotomy can be accomplished.

The finger is inserted through an opening in the tip of the appendage and the valve and its orifice are explored. In a few seconds the pliability of the valve can be ascertained, where the calcium, if any, lies, and where the commissures are. The anterolateral commissure lies at about 11:00 o'clock, the posteromedial at about 5:00 o'clock. The George P. Pilling Son Company and, more recently, Bruno Richter, have been most helpful in developing these guillotine knives. This demonstrates the method of cutting or starting the cut in the anterolateral commissure, and this particular knife can be turned over and used for the posteromedial commissure as well. Usually the knife will be used with the curve the other way and the anterolateral commissure just opened. One little nick is taken in the commissural angle. Here you see what tremendous improvement just one small cut will make. Frequently, if finger pressure alone is relied upon, all that part of the valve lying parallel to the wall of the ventricle will be avoided or missed, so that we prefer to have a hooked blade available at all times. You see now what a marked opening can be produced by just the anterolateral commissurotomy. One small cut may be made on the posteromedial commissure in this fashion and a little additional opening obtained, but we do not carry the cut on this side very far, for otherwise one might incise into the outflow tract of the left ventricle (the intracardiac portion of the aorta) and thereby produce excessive mitral regurgitation.

NEW INSTRUMENT FOR CARDIAC VALVULAR COMMISSUROTOMY

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THE surgical attack on aortic stenosis has long frustrated the cardiovascular surgeon. Transaortic, including transcarotid approaches, have met with significant failure. Earlier attempts via the left ventricle have resulted in some catastrophes due to unguided and blind techniques.

A successful new instrument consisting of three main features is presented.† It contains, first, a beaded guidewire; second, an expanding or dilating triradiate mechanism for aortic commissurotomy; third, an operating control mechanism.

First, an estimate of the external diameter of the aortic valve ring is made by manual palpation. The instrument's control mechanism is then accurately set, so that the open diameter of the dilator will be slightly less than that of the aortic ring. This prohibits overstretching, tearing, or cracking of the aorta at the time the aortic valve commissures are opened.

By passing the beaded wire into the left ventricle, through a small incision under purse-string control, and up into the stenosed aortic valve orifice, a positive guide is then in place as a path for the dilator to follow. This eliminates time-consuming and injurious probing. The triradiate arms of the dilator are wedge-shaped, as seen on cross section, to allow a leading fin on each arm to initiate the parting of the commissures along their fused planes. The dilating head of the instrument is set on a slight swivel, so that it becomes self-aligned to meet the commissures accurately, as the head is passed into the valve orifice. The dilating action is one of commissure parting, not tearing, or cutting. The stretching action at the mid-portion of the leaflet, which can result in fatal tear, is minimized because of the parting action at the commissures.

The anatomic position of the anterior commissure of the aortic valve is nearly always constant, and an external position overlying this point is readily identified in relation to the take-off of the coronary arteries. Thus, in the original passage of the instrument through the ventricle, the anterior dilating arm of the dilator is positioned roughly, but accurately, by merely turning the handle and shaft a few degrees or, as we would say, "lining it up" with the point in question. The swivel feature of the dilating mechanism then accomplishes accurate alignment of the dilator as it fits into the orifice.

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†We are greatly indebted to Major John Shearman Donaldson for the mechanical design and production of this instrument. It may be purchased through George P. Pilling Co., Philadelphia, Pa.

A safety device prohibits opening of the dilator upon entering and withdrawing of the instrument through the ventricular wall.

The position of the acting head of the mechanism is assured by external palpation at the root of the aorta. To date, twenty-three patients have been operated upon, thirteen of the operations being combined with mitral com-

Fig. 1.

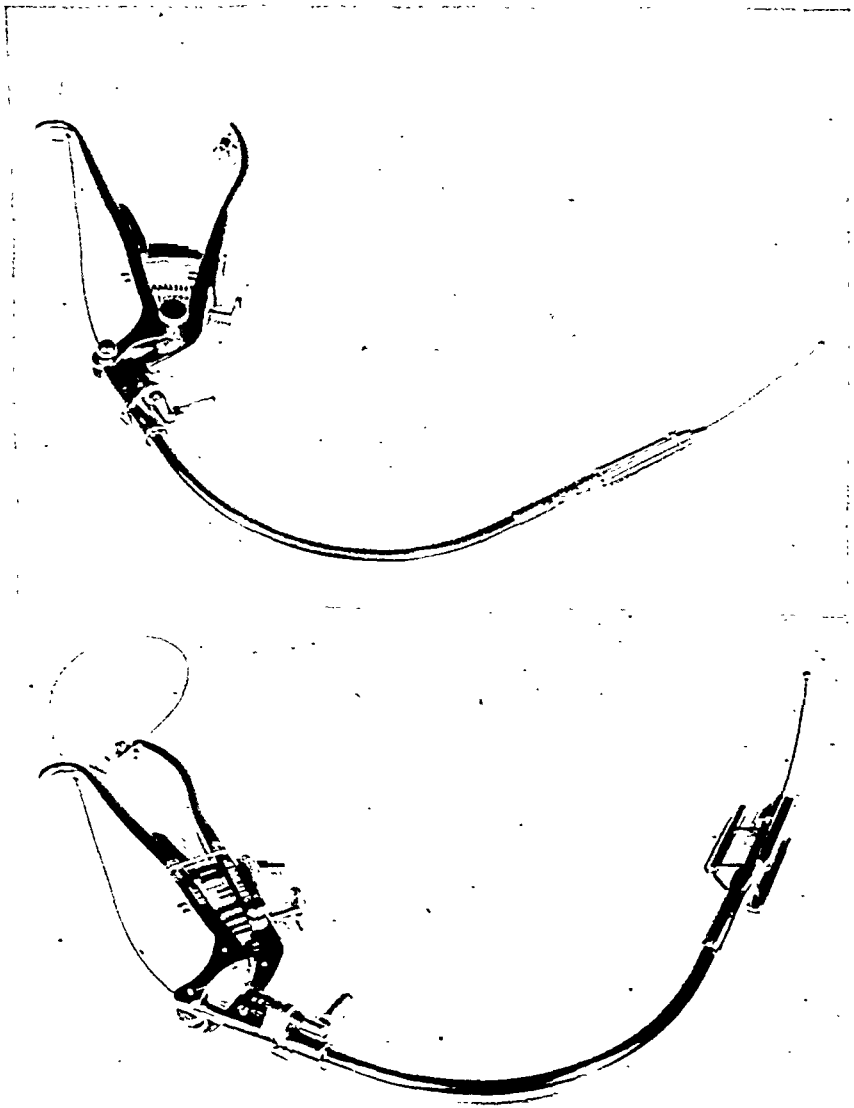


Fig. 2.

Fig. 1.—Aortic dilator (closed.)
Fig. 2.—Aortic dilator (open).

missurotomy, done at the same operation. There has been one operative mortality.

The simplicity of action and the results of this instrument have given us great hope for a more complete attack on pulmonary and mitral valve stenoses. A biradiate dilator attachment has been developed for these two valves, being applicable chiefly in adult cases. The Potts dilator and the Brock dilator are good for the pulmonary stenosis of infants and children, and the instrument presented here offers an excellent means of opening the adult pulmonary valve. The pointed, leading end of the dilator, plus its guidewire, assures accurate and quick engagement.

As for the mitral valve, there are some cases where the finger or instrumental techniques as understood to date, have met with failure, due either to

Fig. 3.

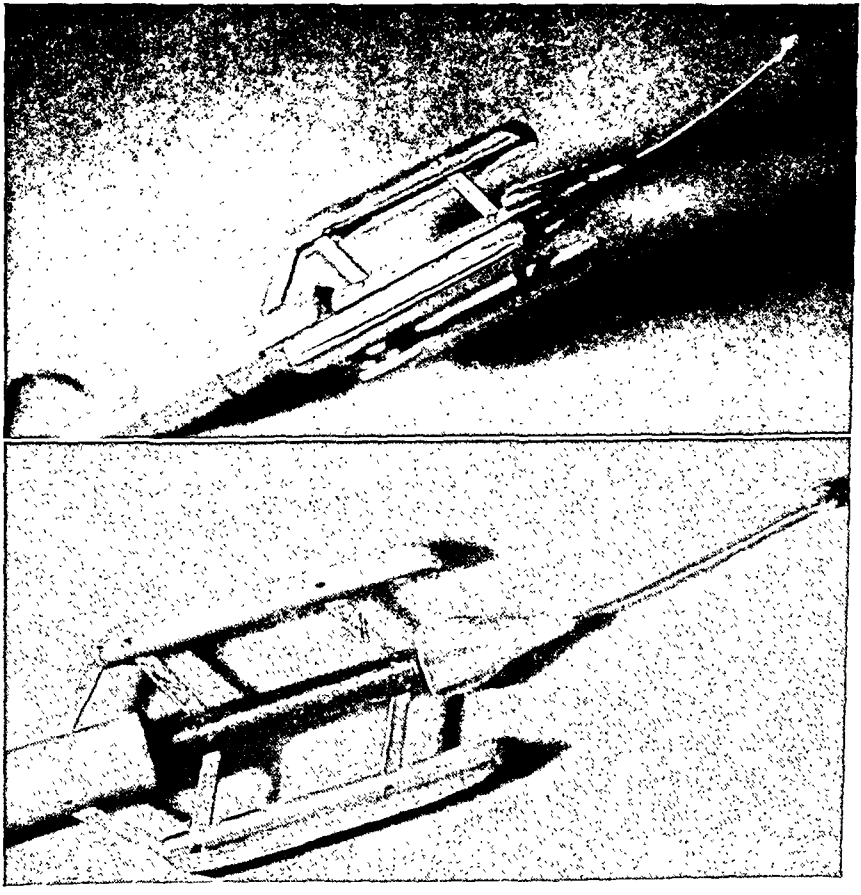


Fig. 4.

Fig. 3.—Triradiate dilator head with guidewire.
Fig. 4.—Biradiate dilator head with guidewire for pulmonary and mitral stenoses.

the thickness of the valve, presence of a tremendous interauricular thrombosis, or a contracted, tiny, inaccessible auricular appendage. In these, biradiate dilatation is offered as a real improvement in mode of attack. Here again, this improvement is noted because the dilating bar is designed to part the commissure, rather than cut or tear it. Also, there is better control in the actual degree or length of opening which can be accomplished.

At present, the instrument is not being used on thin valvular stenoses, as there is a possibility of false splitting of the septal leaflet in mitral stenosis of this type.

PULMONARY RESECTION IN THE TREATMENT OF LUNG TUBERCULOSIS

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THE principle of resection of tuberculous tissue has recently been accepted in the treatment of pulmonary tuberculosis. With the pulmonary resection, only the active focus of tuberculosis is removed, but it is evident that there are quiescent foci spread all over the lung. Following the operation, there can be reactivation of the disease in the remainder of the lung, or dissemination of tuberculosis in new foci.

After the publications of Churchill and Klopstock,[1] Overholt,[3] and others, there was great enthusiasm for this proceduce. However it was pointed out that the results of thoracoplasty were far superior to those of resection; pulmonary resections were followed by more complications, although they were effective in many cases which could not be satisfactorily treated by any other procedure. Naturally, the complications are directly related to the type of lesions for which the operation is indicated.

Sweet,[5] reviewing 63 cases followed over a period of two years, verified that 28 patients (44.4 per cent) had died, 9 showed active lesions (14.3 per cent), and only 26 (41.3 per cent) were clinically well and had negative tests. These patients, who were submitted to 27 lobectomies and 36 pneumonectomies, were operated on before the systematic use of streptomycin.

Overholt[4] demonstrated the influence of anesthesia, of decubitus during the operation, and of corrective thoracoplasty on the results of lung resection in tuberculosis. Jones and Robinson[2] assembled statistics covering operations performed before 1947 from the records of three large surgical clinics. From a total of 343 cases, the morbidity rate was 51 per cent and the mortality rate 28 per cent. These authors, in a series of 123 pulmonary resections, reported complications in 22 per cent of the cases and a mortality of 12.2 per cent. Among 80 patients receiving streptomycin, the morbidity was 12.5 per cent and the mortality 5.7 per cent.

CLINICAL MATERIAL

This report deals with the results of resection for pulmonary tuberculosis observed in a series of 103 patients, performed from February, 1946, through January, 1951. The general statistical data on these patients are shown in Table I.

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TABLE I. GENERAL STATISTICS (PERIOD BETWEEN FEB., 1946, AND JAN., 1951) ON PULMONARY RESECTION IN TUBERCULOSIS

1. Number of patients			103
2. Number of resections			103
Pneumonectomies		53	
Right	25		
Left	28		
Lobectomies		49	
Right upper	24		
Right upper and middle	1		
Right upper and superior Division of right lower	3		
Right lower	6		
Right lower and middle	2		
Left upper	11		
Left lower	2		
Segmental resection		1	
			103
3. Age of patients (15 to 50 yr.)			
15 to 20 yr.	15		
21 to 30 yr.	53		
31 to 40 yr.	24		
41 to 50 yr.	11		
			103
4. Sex			
Males	42		
Females	61		
			103
5. Duration of disease			
Under 1 yr.	32		
1 to 2 yr.	40		
2 to 3 yr.	14		
3 to 4 yr.	4		
4 to 5 yr.	3		
Over 5 yr.	10		
			103
6. Race			
White	89		
Black	9		
Yellow	5		
			103
7. Incidence of positive sputum	96 (93.2%)		

The cases were thus distributed: 53 pneumonectomies, 49 lobectomies, and 1 segmental resection. In 3 cases, we performed at the same time a lobectomy and a segmental resection in another lobe. The pneumonectomies were done on the right side in 25 instances and on the left side in 28 instances.

The lobectomies were as follows:

24 excisions of the right upper lobe
1 excision of both right upper lobe and middle lobe
3 of the right upper lobe and apical segment of the right lower lobe
6 of the right lower lobe
1 of both the right lower lobe and the middle lobe
11 of the left upper lobe
2 of the left lower lobe

In one case, the apicoposterior segment of the left upper lobe was excised.

The youngest patient of this group was 15 years old, and the oldest, 50 years old. The majority of the patients were in the 21-to-30 group; 61 were women and 42 men. Thirty-two per cent of the patients were sick for less than one year. Of the patients, 96 (93.2 per cent) had positive sputum and 7 (6.8 per cent) were negative before the operation. These patients had bronchial stenosis, bronchiectasis, and "filled" tuberculous cavities.

INDICATIONS

In the beginning of this series, our indications were more conservative, choosing the cases in which the operation could offer a lower risk. However, we soon realized that a great number of patients with severe pulmonary lesions could be cured only through pulmonary resection; these patients presented more complications and fewer cures.

The indications under which our patients were operated on are summarized in Table II.

TABLE II. INDICATIONS IN ONE HUNDRED THREE PULMONARY RESECTIONS IN TUBERCULOSIS

1. Persistent lesions under collapse therapy		41
A. Cavity under pneumothorax	14	
B. Exudative lesions under pneumothorax	11	
C. Cavity under extrapleural pneumothorax	4	
D. Postthoracoplasty uncontrolled disease	12	
2. Large cavities of the upper lobe, with evidence of bronchial disease		16
3. Lower lobe cavities (1 case associated with suppurative disease)		10
4. Extensive unilateral tuberculosis*		24
5. Fibrosis, bronchiectasis, and tuberculous tracheobronchitis†		4
6. Upper lobe disease (suitable for thoracoplasty)		3
7. Tuberculoma or "filled" cavities		4
8. Tuberculosis of a single pulmonary segment		1
Total		103

*One patient with bronchialstenosis and negative sputum.
†Two patients with negative sputum.

In the first group are 41 persistent lesions under collapsotherapy. Among them, there are 14 cavities under total pneumothorax and phrenicotripsia; 11 exudative lesions (caseous pneumonia) under pneumothorax and phrenicotripsia; 4 cavities under extrapleural pneumothorax, and 12 residual cavities after pneumothorax.

In the second group are 16 cases of large cavities of the upper lobe, with evident signs of bronchial tuberculosis; many of these cases represented an actual disappearance of the upper lobe.

In a third group are 10 cases of cavities of the lower lobe, all of them persistent after phrenicotripsia.

The fourth group concerns 24 cases of extensive lesions, multilocular, localized in a single lung.

In the fifth group are 4 cases of specific bronchiectasis, bronchial tuberculosis, and more or less pronounced fibrosis of the lung.

In the sixth group we included three cases of excavated upper lobitis; in these cases we had to determine whether the indications were for lobectomy or thoracoplasty.

In the seventh group are 4 cases of cavities with a closed bronchus, "filled" with organizing caseous material. These cases are similar to the cases described as tuberculomas by the American authors.[2]

The eighth group considers a patient in whom the lesions were restricted to a segment of the lung. In this case a segmental resection was performed.

Some of these indications, such as lesions under thoracoplasty, insufflated lesions under pneumothorax, and the cavities of the lower lobe not cured by the common collapsotherapeutic methods, are very well established indications in which the results are generally satisfactory. The other groups of indications were considered as our experience increased. The majority of these cases involve lesions where only resection of that diseased part of the lung could bring about a cure.

Results and postoperative complications of pulmonary resection generally vary according to the type of lesion, and, therefore, to the indication under which the operation was performed.

In Table III are set forth the relations between the operative indications, the postoperative complications, and the final results.

Old opened cavities under pneumothorax are a good indication for resection. Among 14 patients operated upon for this indication, 5 pneumonectomies and 9 lobectomies were performed, and only 2 patients presented postoperative complications, 1 being dissemination and 1 bronchial fistula and empyema. The 12 patients without postoperative complications are clinically cured. Of the other 2, one died and the other still has lesions with positive sputum.

The exudative lesions, as we expected, represent the poorest indication for resection. Also these lesions show poor results no matter what therapeutic method is used. The patients operated on presented severe lesions of the caseous type, which commonly end in death under any of the usual treatments. Of the 11 resections, 9 were pneumonectomies and 2 lobectomies, with the following results:

Four presented disseminations; 5 had reactivation of old foci; 3 developed empyema, and only 2 showed an uneventful postoperative course. Five patients died; 3 are alive but still have lesions and positive sputum; only 3 patients out of 11 are apparently well and have negative sputum.

Two pneumonectomies and 2 lobectomies were performed in a group of 4 patients presenting residual cavities under extrapleural pneumothorax. In the postoperative period there was 1 dissemination; 1 reactivation; 2 bronchial fistulas with empyema; and only 1 patient had no complications. Two patients are dead; 1 is alive but still has lesions and positive sputum; only 1 patient is well and negative from the bacteriologic viewpoint. The patients belonging to this group were operated on in the beginning of this series, when our experience was still small. Although resection under extrapleural pneumothorax might

TABLE III. RESULTS IN RELATION TO THE INDICATION FOR OPERATION IN PULMONARY RESECTION IN TUBERCULOSIS

INDICATIONS	NUMBER	TYPE OF RESECTION		POSTOPERATIVE COMPLICATION					FINAL RESULTS			
									DEATH			
		PNEUMONECTOMY	LOBECTOMY AND SEGMENTAL RESECTION	SPREAD	FLARE-UP	BRONCHIAL FISTULA AND EMPYEMA	EMPYEMA WITHOUT BRONCHIAL FISTULA	NO COMPLICATION	IN DIRECT RELATION TO THE RESECTION	OTHER CAUSES*	APPARENTLY WELL, SPUTUM NEGATIVE	ALIVE WITH DISEASE
Cavity under pneumothorax	14	5	9	1	-	1	-	12	1	-	12	1
Exudative lesions under pneumothorax	11	9	2	4	5	-	3	2	3	2	3	3
Cavity under extrapleural pneumothorax	4	2	2	1	1	2	-	1	2	-	1	1
Postthoracoplasty disease	12	5	7	-	-	3	-	9	1	1	10	-
Large cavity of the upper lobe	16	6	10	4	3	4	1	3	2	4	9	1
Lower lobe cavity	10	-	10	2	2	4	1	1	2	2	6	-
Extensive unilateral tuberculosis	24	24	-	1	5	3	3	13	1	2	18	3
Fibrosis, bronchiectasis, & bronchial tuberculosis	4	1	3	-	1	-	-	3	-	-	4	-
Upper lobe disease suitable for thoracoplasty	3	-	3	1	1	1	-	1	1	1	1	-
Tuberculoma or filled cavity	4	1	3	-	-	2	-	2	-	-	3	1
Tuberculosis of a single pulmonary segment	1	-	1	-	-	-	-	1	-	-	1	-

*See *Mortality*.

sometimes be more difficult technically, our impression is that it should be considered a good indication, offering at present better results.

Also the cavities under thoracoplasty are to be considered a good indication. In a group of 12 patients, 5 pneumonectomies and 7 lobectomies were performed. Bronchial fistulas with empyema occurred in 3 patients; the other 9 presented no complications. From those 12 patients, 2 died and 10 are clinically well. As happened in the other groups studied, some of the deaths are directly traceable to the resections; however, others are not. This fact will be studied later on.

Six pneumonectomies and 10 lobectomies were done in a group of 16 patients with large insufflated cavities of the upper lobe. In some cases, there were also lesions in other lobes, which was the reason for our pneumonectomy. These patients presented the following postoperative complications: 4 disseminations; 3 reactivations; 4 bronchial fistulas and empyema; and 1 empyema without bronchial fistula. Only 3 patients presented no complications. Six patients are dead; 1 is alive with lesions; and the other 9 are clinically cured.

Ten lobectomies were performed on 10 patients with lesions of the lower lobe; there were 2 disseminations; 2 reactivations; 4 bronchial fistulas and empyema; 1 empyema without bronchial fistula; and 1 patient presented no complications. Four patients died and 6 are clinically cured.

Twenty-four patients with extensive lesions, in one lung only, were treated by pneumonectomy, with the following results: 1 dissemination; 5 reactivations; 3 bronchial fistulas and empyema; and 3 cases of empyema without bronchial fistula; the evolution in 13 patients was without postoperative complications. Of these 24 patients, 3 died, 3 are living with lesions, and 18 are clinically cured.

One pneumonectomy and 3 lobectomies were performed on patients with bronchiectasis and bronchial tuberculosis. One case presented reactivation and 3 no complications. All 4 patients are cured.

On 3 patients with lesions of the upper lobe where we could start with thoracoplasty, 3 lobectomies were performed; we had 1 dissemination; 1 reactivation; 1 bronchial fistula and empyema, and 1 patient with no complications. Two patients died and one is cured.

Among 4 patients with "filled" and blocked cavities, we performed 1 pneumonectomy and 3 lobectomies. There were 2 bronchial fistulas and empyema, and 2 patients had no complications. One patient still has lesions and positive sputum, and 3 are clinically cured.

One segmental resection was carried out on a patient with lesions of a single pulmonary segment. There were no postoperative complications and the patient is cured.

COMPLICATIONS

Table IV shows the incidence of the main postoperative complications observed in our patients, and the evolution of the complicated cases.

TABLE III. RESULTS IN RELATION TO THE INDICATION FOR OPERATION IN PULMONARY RESECTION IN TUBERCULOSIS

INDICATIONS	NUMBER	TYPE OF RESECTION		POSTOPERATIVE COMPLICATION					FINAL RESULTS			
									DEATH			
		PNEUMONECTOMY	LOBECTOMY AND SEGMENTAL RESECTION	SPREAD	FLARE-UP	BRONCHIAL FISTULA AND EMPYEMA	EMPYEMA WITHOUT BRONCHIAL FISTULA	NO COMPLICATION	IN DIRECT RELATION TO THE RESECTION	OTHER CAUSES*	APPARENTLY WELL, SPUTUM NEGATIVE	ALIVE WITH DISEASE
Cavity under pneumothorax	14	5	9	1	-	1	-	12	1	-	12	1
Exudative lesions under pneumothorax	11	9	2	4	5	-	3	2	3	2	3	3
Cavity under extrapleural pneumothorax	4	2	2	1	1	2	-	1	2	-	1	1
Postthoracoplasty disease	12	5	7	-	-	3	-	9	1	1	10	-
Large cavity of the upper lobe	16	6	10	4	3	4	1	3	2	4	9	1
Lower lobe cavity	10	-	10	2	2	4	1	1	2	2	6	-
Extensive unilateral tuberculosis	24	24	-	1	5	3	3	13	1	2	18	3
Fibrosis, bronchiectasis, & bronchial tuberculosis	4	1	3	-	1	-	-	3	-	-	4	-
Upper lobe disease suitable for thoracoplasty	3	-	3	1	1	1	-	1	1	1	1	-
Tuberculoma or filled cavity	4	1	3	-	-	2	-	2	-	-	3	1
Tuberculosis of a single pulmonary segment	1	-	1	-	-	-	-	1	-	-	1	-

*See Mortality.

sometimes be more difficult technically, our impression is that it should be considered a good indication, offering at present better results.

Also the cavities under thoracoplasty are to be considered a good indication. In a group of 12 patients, 5 pneumonectomies and 7 lobectomies were performed. Bronchial fistulas with empyema occurred in 3 patients; the other 9 presented no complications. From those 12 patients, 2 died and 10 are clinically well. As happened in the other groups studied, some of the deaths are directly traceable to the resections; however, others are not. This fact will be studied later on.

Six pneumonectomies and 10 lobectomies were done in a group of 16 patients with large insufflated cavities of the upper lobe. In some cases, there were also lesions in other lobes, which was the reason for our pneumonectomy. These patients presented the following postoperative complications: 4 disseminations; 3 reactivations; 4 bronchial fistulas and empyema; and 1 empyema without bronchial fistula. Only 3 patients presented no complications. Six patients are dead; 1 is alive with lesions; and the other 9 are clinically cured.

Ten lobectomies were performed on 10 patients with lesions of the lower lobe; there were 2 disseminations; 2 reactivations; 4 bronchial fistulas and empyema; 1 empyema without bronchial fistula; and 1 patient presented no complications. Four patients died and 6 are clinically cured.

Twenty-four patients with extensive lesions, in one lung only, were treated by pneumonectomy, with the following results: 1 dissemination; 5 reactivations; 3 bronchial fistulas and empyema; and 3 cases of empyema without bronchial fistula; the evolution in 13 patients was without postoperative complications. Of these 24 patients, 3 died, 3 are living with lesions, and 18 are clinically cured.

One pneumonectomy and 3 lobectomies were performed on patients with bronchiectasis and bronchial tuberculosis. One case presented reactivation and 3 no complications. All 4 patients are cured.

On 3 patients with lesions of the upper lobe where we could start with thoracoplasty, 3 lobectomies were performed; we had 1 dissemination; 1 reactivation; 1 bronchial fistula and empyema, and 1 patient with no complications. Two patients died and one is cured.

Among 4 patients with "filled" and blocked cavities, we performed 1 pneumonectomy and 3 lobectomies. There were 2 bronchial fistulas and empyema, and 2 patients had no complications. One patient still has lesions and positive sputum, and 3 are clinically cured.

One segmental resection was carried out on a patient with lesions of a single pulmonary segment. There were no postoperative complications and the patient is cured.

COMPLICATIONS

Table IV shows the incidence of the main postoperative complications observed in our patients, and the evolution of the complicated cases.

TABLE IV. POSTOPERATIVE COMPLICATIONS* FROM PULMONARY RESECTION IN TUBERCULOSIS

TYPE OF RESECTION	NUMBER	SPREAD	FLARE-UP REACTIVATION	BRONCHIAL FISTULA AND EMPYEMA	EMPYEMA WITHOUT BRONCHIAL FISTULA	MISCELLANEOUS†	NO COMPLICATIONS
Pneumonectomy	53	6 (11.3%)	11 (20.7%)	7 (13.2%)	7 (13.2%)	3 (5.6%)	26 (49%)
Lobectomy and segmental resection	50	8 (16%)	7 (14%)	13 (26%)	1 (2%)	2 (4%)	22 (44%)
Total	103	14 (13.5%)	18 (17.4%)	20 (19.4%)	8 (7.7%)	5 (4.8%)	48 (46.6%)
Final Results in Relation to Postoperative Complication							
Apparently well, sputum negative		3 (21.4%)	6 (33.3%)	8 (40%)	4 (50%)	—	48 (100%)
Death		9 (64.2%)	6 (33.3%)	7 (35%)	3 (33.3%)	5 (100%)	—
Alive with disease, sputum positive		2 (14.2%)	6 (33.3%)	5 (25%)	1 (12.5%)	—	—

*Some patients presented more than one complication.
†Transfusion accident, pulmonary embolus, intestinal hemorrhage, cardiac failure.

TABLE V. PULMONARY RESECTION IN TUBERCULOSIS

COMPLICATIONS	TOTAL (103 RESECTIONS)				UNTIL DECEMBER 1948 (27 CASES)				JAN. 1949 TO JAN. 1951 (76 CASES)			
	INCIDENCE		MORTALITY		INCIDENCE		MORTALITY		INCIDENCE		MORTALITY	
	NUMBER OF CASES	PER CENT	NUMBER OF CASES	PER CENT	NUMBER OF CASES	PER CENT	NUMBER OF CASES	PER CENT	NUMBER OF CASES	PER CENT	NUMBER OF CASES	CENT PER
Dissemination	14	13.5	9	8.7	8	29.2	5	18.5	6	7.8	4	5.2
Reactivation of old foci	18	17.4	6	5.8	4	14.8	4	14.8	14	18.4	2	2.6
Bronchial fistula and empyema	20	19.4	7	6.8	10	37.0	4	14.8	10	13.1	3	3.9
Empyema only	8	7.7	3	2.9	3	11.1	2	7.4	5	6.5	1	1.3

The most severe complication was the dissemination of the tuberculous process. Its incidence was 13.5 per cent (14 cases) in the entire series, corresponding to 11.3 per cent (6 cases) for the pneumonectomies and 16 per cent (8 cases) for the lobectomies. Of the 14 patients who presented dissemination, only 3 (21.4 per cent) are at this time clinically cured with negative sputum; 9 patients (64.2 per cent) died and 2 (14.2 per cent) still present lesions and positive sputum.

Reactivation of old established foci was observed in 18 cases (17.4 per cent). After the resection, these patients had negative sputum for some time, and then showed exacerbation of foci formerly inactive. This reactivation may or may not be related to the resection. In some cases where resection was not followed by a corrective thoracoplasty, there was an exaggerated expansion of the remaining part of the lung, which may be the cause of the reactivation. However, in other cases, reactivation of old foci took place without apparent cause, as frequently occurs with tuberculous patients. The reactivation of old foci was more frequent in the pneumonectomy group (11 cases, 20.7 per cent) than in the lobectomy group (7 cases, 14 per cent). Of the 18 cases, 6 are cured (33.3 per cent), 6 are dead (33.3 per cent), and 6 still present lesions and positive sputum (33.3 per cent).

Empyema and bronchial fistula was the most frequent complication. Twenty cases were observed (19.41 per cent) consisting of 7 (13.2 per cent) pneumonectomies and 13 (26 per cent) lobectomies. Of these patients, 8 are cured (40 per cent); 7 died (35 per cent), and 5 (25 per cent) still have lesions and positive sputum.

Empyema without bronchial fistula was observed in 8 cases (7.7 per cent), 7 after pneumonectomies (13.2 per cent), and 1 (2 per cent) after lobectomy. Of these patients, 4 are cured (50 per cent); 3 died (33.3 per cent), and 1 (12.5 per cent) still has lesions and is bacteriologically positive.

Other complications indirectly related to the resection were observed in 5 cases (4.8 per cent). These were transfusion accidents, pulmonary embolus, intestinal hemorrhage, cardiac insufficiency, and 1 case of sudden death. These patients all died.

Of the entire series, only 48 patients (46.6 per cent) had no complications, 26 after pneumonectomies (49 per cent) and 22 after lobectomies (44 per cent). All of these patients are cured.

Postoperative complications presented a greater incidence in the early cases of our series, and at the moment they are less frequent. In the first 27 patients operated on, from February, 1946 to December, 1948, streptomycin was not used. It has been proved by various authors that this antibiotic decreases the number of complications and the mortality when administered during the pre- and postoperative periods.

Following 1948, based on our previous experience, we followed a more careful surgical technique; we systematically used the prone position, and the patients were prepared by the administration of 1.0 gr. streptomycin daily for fifteen days. As can be observed in Table V, complications from then on were less frequent and mortality correspondingly lower. Dissemination, which in

the early period represented an incidence of 29.2 per cent, with a mortality of 18.5 per cent, fell off to 7.8 per cent and 5.2 per cent, respectively. The same is true of the bronchial fistulas, in which cases the incidence decreased from 37 to 13.1 per cent and the mortality from 14.8 to 3.9 per cent. Empyema without bronchial fistula was also less frequent; its incidence was reduced from 11.1 to 6.5 per cent and the mortality from 7.4 to 1.3 per cent. Regarding the reactivation of old foci, it was noted that the incidence increased from 14.8 to 18.4 per cent, but the corresponding mortality decreased from 14.8 to 2.6 per cent. In 76 patients operated upon since January, 1949, there have been 40 (52.9 per cent) without postoperative complications, while in the previous series only 8 patients out of 27 (29.1 per cent) presented a postoperative evolution without complications.

TABLE VI. MORTALITY IN PNEUMONECTOMY FOR TUBERCULOSIS

Number of resections	53
Postoperative fatalities (1 month)	4
Late fatalities (1 to 25 months)	8
Causes of death:	
1. Spread	3
2. Flare-up of old lesions*	2
3. Empyema and toxemia	2
4. Bronchial fistula, blockage of airway and asphyxia	1
5. Transfusion reaction	2
6. Pulmonary embolus	1
7. Cardiac failure	1
Deaths directly connected to the resection	6 (11.3%)
Deaths indirectly connected to the resection	4 (7.5%)
Deaths by new progressive lesions in apparently well patients	2 (3.7%)

*Apparently well with negative sputum 2 and 12 months after surgery.

MORTALITY

In Tables VI and VII are summarized all deaths observed during the entire follow-up period for these patients, that is, from nine months to five and one-half years. Many deaths are not directly related to the surgical process. Some were caused by accidents or intercurrent diseases; others were due to the evolution of new foci of tuberculosis in patients clinically cured of the lesions for which the pulmonary resections were performed. Among the 53 pneumonectomies performed, there were 6 (11.3 per cent) deaths directly related

TABLE VII. MORTALITY IN LOBECTOMY AND SEGMENTAL RESECTION FOR TUBERCULOSIS

Number of resections	50
Postoperative fatalities (1 month)	3
Late fatalities (1 to 30 months)	10
Causes of death:	
1. Spread	6
2. Flare-up of old lesions*	4
3. Empyema and toxemia	1
4. Intestinal hemorrhage	1
5. Suddenly (cause undetermined)	1
Deaths directly connected to the resection	7 (14%)
Deaths indirectly connected to the resection	2 (4%)
Deaths by new progressive lesions in apparently well patients	4 (8%)

*Apparently well with negative sputum 2, 4, and 30 months after surgery.

to the operation and caused by postoperative complications—3 disseminations, 2 empyemas, and 1 opening of the bronchus in the immediate postoperative period followed by aspiration of the hemothorax. Four other deaths were indirectly related to the pneumonectomy (7.5 per cent), 2 of these deaths being due to transfusion accidents, 1 to pulmonary embolism, and 1 to cardiac insufficiency. Two patients (3.7 per cent) succumbed on account of the evolution of new pulmonary lesions, after they had shown negative sputum for two and twelve months, respectively.

The causes of death were also divided into three groups concerning lobectomies (Table VIII). In the first group, 7 deaths (14 per cent) were directly related to the resection—6 disseminations and 1 empyema and toxemia. In 2 cases (4 per cent) death was indirectly related to the surgical procedure and in 1 instance followed hemorrhage in intestinal lesions; the other patient died suddenly from an unknown cause. In the third group, four patients (8 per cent) died as a result of new lesions, after periods of two, four, and thirty months with negative sputum tests.

TABLE VIII. RESULTS (FOLLOW-UP, NINE MONTHS TO FIVE AND ONE-HALF YEARS) IN PULMONARY RESECTION IN TUBERCULOSIS

	PNEUMONECTOMY (53)	LOBECTOMY AND SEGMENTAL RESECTION (50)	TOTAL (103)
Clinically well and negative	34 (64.1%)	34 (68%)	68 (66%)
Radiologic lesions and positive	7 (13.2%)	3 (6%)	10 (9.7%)
Deaths directly connected to the resection	6 (11.3%)	7 (14%)	13 (12.6%)
Deaths due to other causes	6 (11.3%)	6 (12%)	12 (11.6%)

RESULTS

Table VIII summarizes total results observed in 103 cases. Sixty-eight patients (66 per cent) are clinically cured, with negative sputum, and are being observed for a period of from nine months to five and one-half years. Among these cured cases, there are 34 pneumonectomies and 37 lobectomies, corresponding to a percentage of cure for these two operations of 64.1 per cent and 68 per cent, respectively.

From a total of 103 patients, 10 (9.7 per cent) still present radiologic lesions and positive bacilloscopy. Among these patients, 7 had pneumonectomies (13.2 per cent) and 3 lobectomies (6 per cent).

Thirteen patients died (12.6 per cent) as a direct result of the resection, 6 of them being pneumonectomies and 7 lobectomies. The mortality was 11.3 per cent in the case of the pneumonectomies and 14 per cent for the lobectomies. Twelve other patients (11.6 per cent) died during this period of observation, due to causes indirectly related to the resection, with a percentage of 11.3 per cent and 12 per cent, respectively, for the 6 pneumonectomies and 6 lobectomies.

DISCUSSION

The statistical data gathered in this series enable us to analyze the problem of pulmonary resection in tuberculous patients. It must be kept in mind that

the first patients were operated on before the systematic use of streptomycin and therefore presented a great number of complications and deaths. The situation is now improved through the evolution of a better technique and the experience acquired in the matter, and a large number of the patients operated on have an evolution without complications.

Results are directly related to the operative indications. The best results were observed in open cavities under pneumothorax; in residual lesions after thoracoplasty; in cavities of the lower lobe; in fibrotic lesions without cavities and with specific bronchiectasis; and in "filled" and blocked cavities due to the closure of the drainage bronchus. In these cases, pulmonary resection offered a satisfactory number of patients clinically cured, accompanied by negative bacilloscopy. Also it was observed that the number of postoperative complications was less in these groups of patients.

In patients with extensive lesions localized in one lung, results were also very gratifying. All of these cases involved extensive lesions of long standing, well established and with little exudation. One technical detail that probably favored results in this group was the possibility of closing the bronchus of the diseased lung by means of special tubes and balloons, making it possible to perform the pneumonectomy without the danger of dissemination of tuberculosis to the other lung. This is not always possible when performing a lobectomy.

The poorest results in our series were observed among the patients with exudative lesions and caseous pneumonias under pneumothorax. These patients, with less established lesions, presented a great number of postoperative complications and a high mortality rate.

When the tuberculous process is limited to a single pulmonary segment, segmental resection generally offers good results. In this series of patients, operated on up to January, 1951, we had only one case of segmental resection. Since January, 1951, there have been other cases, which will be discussed in a later communication.

From a technical standpoint, many details could be discussed, as pulmonary resection in tuberculosis requires special care not necessitated in resections performed for other pulmonary diseases. Whenever possible, the bronchus corresponding to the part of the lung to be removed was blocked. This blockage was done by means of tubes carrying balloons, or double-lumen tubes. The Overholt prone position was always used, and we always took care to ligate the bronchus first. Whenever possible, before, during, or after the resection, a corrective thoracoplasty was performed. In some cases, the large distention of the remaining part of the lung was prevented by the use of pneumothorax. We do consider it important to protect the remaining lung. Of our patients, 17.4 per cent presented reactivation of old lesions, after a variable period of negative bacilloscopy, after the resection. In these patients, no protective thoracoplasty was performed to offset the hyperdistention of the lung. This is especially dangerous after pneumonectomies, due to the shifting of the mediastinum. In 20.7 per cent of our pneumonectomy patients on whom no posterior thoracoplasty was carried out, there developed reactivation of old foci within the remainder of the lung, after the absorption of the hemothorax.

TABLE IX. RESULTS (FOLLOW-UP FROM NINE MONTHS TO FIVE AND ONE-HALF YEARS) IN PULMONARY RESECTION IN TUBERCULOSIS

	TOTAL (103 CASES)	UNTIL DECEMBER 1948 (27 CASES)	JAN. 1949 TO JAN. 1951 (76 CASES)
Clinically cured	68 (66.0%)	13 (48.1%)	55 (72.3%)
Radiological lesions and sputum positive	10 (9.7%)	2 (7.4%)	8 (10.5%)
Mortality directly connected to the resection	13 (12.6%)	6 (22.2%)	7 (9.2%)
Mortality due to other causes, including new foci of tuberculosis	12 (11.6%)	4 (14.8%)	8 (10.5%)

Late results of pulmonary resections in tuberculosis remain precarious. A mortality of 12.6 per cent and a high morbidity of 53.4 per cent due to late postoperative complications would indicate this method to be inferior to other procedures of surgical collapsotherapy. Only 66 per cent of the patients of this series are clinically cured, whereas we know that the percentage of cures by other procedures can be higher. We must recall, however, that the results of the resections show constant improvement with the experience acquired, the latest patients in our series presenting fewer complications and a greater number of cures. From this point of view, we can divide our 103 cases into two groups (Table IX).

The first group includes 27 patients operated on up to and including December, 1948; these patients did not receive streptomycin routinely in the pre- and postoperative periods, and the surgical procedure in these cases did not include certain technical details which have now been introduced. Of these patients, 48.1 per cent are clinically cured; 7.4 per cent still present radiological lesions and positive sputum; 22.2 per cent died from causes directly related to the resection; and 14.8 per cent died from other causes, including those deaths due to new foci of tuberculosis.

In the second group are 76 patients operated on from January, 1949, to January, 1951. In this group, the patients all received streptomycin routinely, and there were 72.3 per cent of the patients clinically cured; 10.5 per cent with radiological lesions and positive sputum; a mortality of 9.2 per cent due to causes directly related to the resection; and 10.5 per cent of deaths due to other causes.

SUMMARY

1. One hundred three cases of pulmonary resections for the treatment of pulmonary tuberculosis were studied. These patients were operated on from February, 1946, through January, 1951, there being 53 pneumonectomies, 49 lobectomies, and 1 segmental resection. In a group of 76 patients operated on after December, 1948, streptomycin was administered routinely pre- and postoperatively. These patients showed better results than did those operated on earlier. The following data refer to these 76 patients.

2. The principal postoperative complications were:

(a) *Dissemination* in 7.8 per cent of the cases, more commonly observed after lobectomies than after pneumonectomies;

(b) *Reactivation* of old established foci in 18.4 per cent of the cases, more frequently found in pneumonectomies than in lobectomies;

(c) *Bronchial fistula and empyema* in 12.1 per cent of the cases, more often following lobectomies;

(d) *Empyema without bronchial fistula* in 6.5 per cent of the cases, more commonly after pneumonectomies.

3. Of the patients, 9.2 per cent died due to causes directly connected with the resection; mortality was less in pneumonectomies than in lobectomies. Of the patients, 10.5 per cent died due to causes indirectly related to the resection; included in this group are deaths due to new foci of tuberculosis after a period of negative bacilloscopy.

4. Of the patients, 72.3 per cent are clinically cured and have negative sputum, and 10.5 per cent still present radiological lesions with positive sputum.

5. The patients who showed the smallest number of postoperative complications and the largest number of cures were operated upon under the following indications: open cavities under pneumothorax, residual lesions after thoracoplasties, cavities of the lower lobes, and fibrotic lesions without cavities and with bronchiectasis and "filled" and blocked cavities.

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A SIMPLE TEST OF PULMONARY FUNCTION EMPLOYING THE OXIMETER

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IN RESPONSE to a constantly increasing demand for means of evaluating pulmonary function in patients with pulmonary disease, a great many testing procedures have been developed. The ventilatory mechanics of the lungs may now be adequately evaluated by measurements of vital capacity, maximum breathing capacity, breathing reserve, and a study of the breathing pattern.[1] Evaluations of the efficiency of gas exchange in the lungs are not as readily made.[2] Intensive analyses of pulmonary function of the type described by Riley and Cournand[3] are of great value in selected cases, but such procedures are well beyond the scope of routine tests to be employed in the average institution.

The oximeter has come to be recognized as a simpler means of obtaining information as to blood gas exchange, and many applications of this instrument have been described. Because of the obvious importance of the speed with which gases can be equilibrated between the inspired air and the blood, several have studied the time required for the change in hemoglobin saturation between air and oxygen breathing.[4-6] Unfortunately, tests of this type concern themselves with the flat portion of the hemoglobin dissociation curve where significant changes in blood oxygen tension correspond with changes of only a few percentage points in saturation, a circumstance demanding extreme accuracy of the measuring instrument. Inherent limitations in the method of ear oximetry[7] render such accuracy difficult, if not impossible, to attain. It would be much more desirable to work at levels of hypoxia where the steeper slope of the dissociation curve would minimize the significance of small instrumental errors. In addition, direct analytical methods have clearly documented the value to be derived from studying patients under conditions of mild hypoxia as well as under normal conditions of oxygenation.[8] We have therefore developed a method which offers the advantages of simplicity combined with a relatively high degree of accuracy to measure the rapidity with which a patient can resaturate the blood hemoglobin from the hypoxic condition. Aside from the oximeter, it requires no equipment other than that which is available in any hospital.

Apparatus.—This test requires a single-scale compensating oximeter of the Millikan type.* To avoid the inaccuracies of saturation changes over a very narrow range, a 10 per cent change in saturation was selected as the reference

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*We have employed oximeters manufactured by Coleman Instruments, Inc., Maywood, Ill., and by Waters Conley Co., Rochester, Minn. In general, we have found the Coleman instrument more satisfactory for routine testing purposes, although the Waters Conley instrument is required if deeply pigmented Negroes are to be tested.

index. Some degree of instability in the saturation readings may result from minor fluctuations in the excitor lamp voltage. We have therefore introduced a voltmeter with an easily read 3 inch scale into this circuit, together with a series resistance for adjusting the voltage. By setting this voltage slightly below the maximum obtainable, drifts in the battery voltage as indicated on the meter can be compensated by adjusting the resistance and thereby hold the intensity of the excitor lamp constant. This arrangement has the further advantage that with the occasional patient whose skin is too delicate to tolerate the normal heat of the lamp, a slightly lower voltage may be selected and thereby eliminate discomfort for the patient.

In addition to the oximeter, the test requires use of a spirometer equipped with a CO_2 absorber of the type used for basal metabolism recording, and some form of timing device. The respirometer as described by Cournand[1] is particularly well suited to this procedure, but any basal metabolism recording machine having a capacity of 6 liters or more may be used. It is desirable, though not essential, to have the cutoff valve of the spirometer built into the mouthpiece connector at the patient end of the breathing tubes, so as to be able quickly to switch the patient from spirometer breathing to room air breathing. Instruments with the cutoff valve located in the base of the spirometer would require removing the mouthpiece to accomplish this switch unless a correction factor is introduced for the dead space of the breathing tubes. Similarly, to facilitate changing the gas mixture in the spirometer without removing the mouthpiece from the patient, a pair of large three-way valves have been introduced into the breathing tubes through which the spirometer may easily be flushed and filled with room air. For timing purposes, any clock or watch with a sweep second hand may be used, but a stop watch or second indicator of the metronome type is preferable, since it permits the operator to keep his eyes fixed on the oximeter while the resaturation time is measured.

Procedure.—The patient is instructed to lie down and relax upon a suitable cot. The oximeter is turned on and adjusted, and the earpiece applied to the patient. A ten-minute interval must then elapse to permit warming of the ear and maximal dilation of the blood vessels.[9] We take advantage of this interval by using it to obtain vital statistics and a brief history from the patient. The patient is reassured as to the simple nature of the test and impressed with the fact that his only duty will be to relax and breathe naturally into the spirometer until the test is completed.

At the end of the warm-up period, the spirometer is filled with pure oxygen, the mouthpiece and noseclip applied, and the patient begins to breathe into the spirometer. For some edentulous patients, or others who find this method quite uncomfortable, a face mask may be substituted, providing the connections to the spirometer do not significantly increase the dead space. The voltage reading in the excitor lamp circuit is checked at intervals throughout the test, and kept accurately at the predetermined value. We usually run the lamp at 5.7 volts instead of the 6 volt maximum to permit adequate adjustment for battery drifts.

Unless severe functional deficiencies are present, the oximeter will usually be well stabilized at the end of three minutes of oxygen breathing at a level which, by adjustment if necessary, should read 100 per cent saturation on the oximeter scale. If at this time an upward drift of the oximeter persists, oxygen breathing is continued until a 100 per cent reading is maintained. The valve is then turned to cut the patient off from the spirometer and allow him to breathe room air. Within three to four minutes most patients will show a drop to a stable saturation level which is read on the oximeter and recorded as the *basal saturation*. With severe deficiency a longer time lapse may be required to achieve stabilization.

While waiting for stabilization, the spirometer is emptied and then filled and emptied three times with room air. With three-way valves in the breathing tubes this may be accomplished without disturbing the patient, otherwise it will be necessary to remove the mouthpiece in order to flush the spirometer. The spirometer bell is then filled with about 6 liters of *room air*. The exact volume required will vary with the volume of the dead space of the particular apparatus employed, but the total volume should be sufficient to allow the patient to consume about 1.5 liters of oxygen without danger of exhausting the gas volume in the spirometer bell on inspiration. An unnecessarily large initial volume of air in the spirometer serves only to prolong the test. After the spirometer has been flushed and properly filled with air and the patient stabilized at his basal saturation level, he is turned back for spirometer breathing.

It will be apparent that as the patient rebreathes from this air-filled spirometer, his oxygen consumption will progressively lower the oxygen tension in the system. For the first three to four minutes the hemoglobin saturation will fall very slowly. Then as the oximeter reading drops below 90 per cent, a progressively more rapid fall is observed. This fall is frequently cut short for a brief period by mild reflex hyperpnea. In any case, the oximeter is watched closely until the reading drops to 80 per cent saturation. The patient is then immediately cut off from the spirometer and switched to breathing room air. If the apparatus does not have a valve at the mouthpiece, the mouthpiece must be removed at this point to achieve immediate conversion to room air breathing. *The time is accurately measured for the return of the saturation from 80 to 90 per cent.* This time is recorded as the *resaturation time*.

This reading terminates the test unless a deficient resaturation time of more than twenty seconds is recorded. In that case we have found it advisable to recheck the setting of the oximeter to make certain that there is no instrumental error. This is accomplished by refilling the spirometer with pure oxygen and turning the patient back for spirometer breathing for another two to three minutes. Proper instrument standardization will be indicated by a return of the oximeter reading to 100 per cent. We regard a recheck value of over 98.5 per cent with oxygen breathing as evidence of a valid test; a recheck which stabilizes below this level with oxygen breathing indicates significant instrumental or technical error and the test should be repeated. Instrumental drifts of the order of 1 per cent between the beginning and end of the test, however, do not significantly alter the validity of the resaturation time determination.

In carrying out this test, it is obviously essential to maintain efficient absorption of CO_2 in the spirometer circuit at all times in order to exclude any complications due to hypercapnea. This necessitates changing the soda-lime absorber after every ten to fifteen tests, depending upon the capacity of the absorber.

Normal Values.—Many studies have been made of the basal hemoglobin saturation in normal adults. Although the results have shown some variation, most recent work has placed the normal saturation level at somewhere between 96.0 to 98.5 per cent. Thus Wood and associates[9] found in 35 normal adults an average value of 96.7 ± 0.3 per cent, while in 41 subjects Ferguson and coworkers[10] obtained an average of 96.0 ± 0.17 per cent. Greifenstein and associates[11] cited normal values in their laboratory of 97.4 ± 2.1 per cent. Drabkin and Schmidt[12] studied 5 normal subjects, and found all values to be above 98 per cent. Barach and Steiner[13] studied 8 normal subjects and 11 patients with

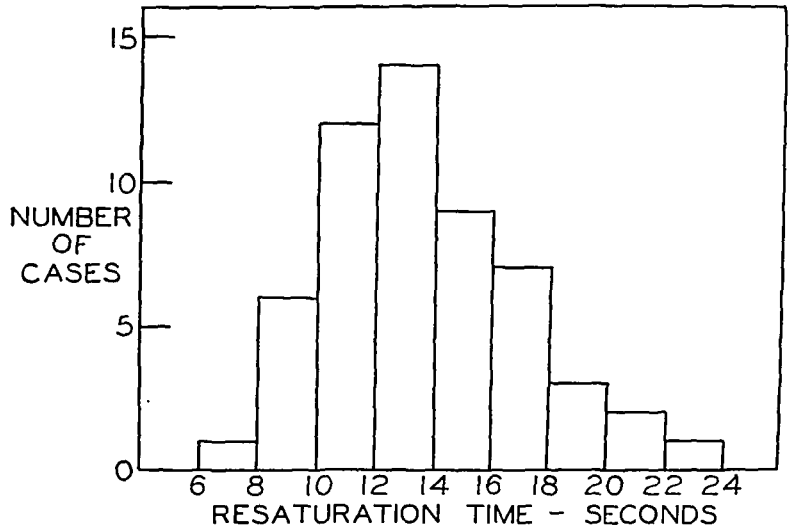


Fig. 1.—Resaturation time values (80 to 90 per cent) measured on 55 normal adults.

coronary artery disease, and in no case found a basal saturation below 96 per cent. In view of these findings, and on the basis of our own experience, we have set 96.0 per cent as the lower limit of what we regard as a definitely normal value for the basal saturation. Obviously the selection of a normal limit as high as this does not warrant the assumption that patients who fall slightly below this limit must necessarily have a pathologic deficiency. Nevertheless it indicates that a subnormal function level is being maintained which, when correlated with other findings, may appear to have clinical significance.

Normal values for the resaturation time recorded in this test were obtained from tests on 55 normal adults in good health who had no history of pulmonary or cardiac disease. This group was composed of 18 males and 37 females with an average age of 23 years, ranging from 17 to 46 years. No significant differences

were observed between the sexes or between the younger and older individuals in the resaturation times recorded. The results of this study are shown in Fig. 1. The mean resaturation time was 13.4 seconds with a standard deviation of ±3.4 seconds. On this basis a resaturation time of 20 seconds, which is approximately two standard deviations from the mean, has been selected as the upper limit of normal. Resaturation times between 21 and 30 seconds are considered suggestive of some pathologic deficiency and values above 30 seconds are considered to indicate definite pulmonary deficiency.

Results of the Test in Patients with Known Pulmonary Disease.—Table I summarizes the readings obtained upon 300 hospitalized patients with pulmonary tuberculosis. No specific selection was involved except for the exclusion of patients who showed clinical evidence of cyanosis. It should be emphasized that this test is designed specifically to detect unrecognized cases of deficiency in pulmonary gas exchange and is not applicable to patients with definite cyanosis in whom the basal saturation will be well below the 90 per cent level.

TABLE I. DISTRIBUTION OF TEST RESULTS ON 300 TUBERCULOUS PATIENTS

RESATURATION TIME (SECONDS)	BASAL-SATURATION (PER CENT)					TOTAL
	100-98.0	97.9-96.0	95.9-94.0	93.9-92.0	91.9-90.0	
Less than 21	17	123	28	1	0	169
21-30	1	29	17	1	0	48
31-60	2	22	13	6	0	43
61-120	0	6	12	6	0	24
More than 120	0	2	5	3	6	16
Total	20	182	75	17	6	300

In evaluating the results shown in Table I, consideration must be given to the pathologic physiology that may be involved in these cases. Riley and Cournand[3] have stressed the importance of maintaining a normal ventilation/perfusion ratio in the lung alveoli. Ventilation may be deficient due to a purely quantitative reduction in the volume of ventilation for either local alveoli or an entire lung. This would be observed in such conditions as therapeutic collapse, severe pleural thickening preventing adequate expansion of the lung, and to varying degrees in many types of infiltrative processes. Such a pure *ventilatory* defect would obviously result in inadequate aeration of the blood perfusing the alveolar capillaries and thus act to lower the basal saturation. Nevertheless, when confronted with a sudden change in the inspired oxygen tension, there is no barrier to rapid gas diffusion in such lungs and therefore the resaturation time would be influenced to only a minor degree.

In contrast, ventilation may be interfered with in a qualitative manner by imposing a barrier to the diffusion of gases between the inspired air and the lung alveoli. This defect is observed classically in emphysema with its large dead spaces interfering with rapid diffusion, and would also be observed to varying degrees in the presence of any fluid barrier to gas diffusion as occurs with edema and many exudative types of disease. These *diffusion* defects will tend to lower the alveolar oxygen tension somewhat. However, since hemoglobin

saturation does not drop significantly until there has been a marked reduction in the alveolar oxygen tension, the basal saturation will not be lowered significantly until the defect becomes severe. However, when presented with the problem of resaturation from the hypoxic condition, a patient with a barrier to efficient gas diffusion should exhibit a significant prolongation of his resaturation time.

To evaluate the possibility of differentiating these conditions with the oximeter test employed here, oximetry findings were compared with a careful analysis of breathing patterns for evidence of functional emphysema. As described by Cournand,[1] breathing patterns which show abnormal prolongations of deep expirations, inability to maintain a rapid rate of breathing, and a tendency for the expiratory position to ride up toward the inspiratory position with successive deep or rapid respirations were considered as mechanical evidence of significant functional emphysema which might be expected to be associated with diffusion defects. Of the 32 patients in Table I who showed saturation times greater than 30 seconds but basal saturations above 96.0 per cent, all 32 showed marked evidence of emphysema in their respiratory patterns. None of these patients were found to have had any therapeutic collapse. These findings are in accord with what would be anticipated in the presence of a diffusion defect.

On the other hand, patients with basal saturations below 96 per cent but with resaturation times of less than 21 seconds should represent those with simple ventilation deficiencies. Actually 25 of the 29 patients in this latter group were found to have had therapeutic collapse of one or both lungs when the test was made. Only 7 patients exhibited respiratory patterns which we would consider to be suggestive of some emphysematous disturbance, and in none of these was the condition marked.

In the remaining deficient groups, no consistent correlation was observed between clinical findings, respiratory patterns, and oximetry readings which would justify the use of the oximetry readings to differentiate the nature of the pulmonary disturbance. On this basis, of the total of 160 patients who were considered to have some deficiency in their oximetry, 99 or 62 per cent were found to have deficiencies of an apparently mixed type which could not be differentiated further, while in 38 per cent the deficiency could be identified as being either of the ventilatory or the diffusion types. In terms of the total tuberculous population of 300 patients tested, 47 per cent had no oximetric deficiency, 33 per cent had deficiencies of a mixed type, and 20 per cent had deficiencies which could be differentiated as either ventilatory or diffusion in nature.

Reproducibility.—The reliability of any test must be established in terms of its ability to give equivalent values when the same subject is retested. We have found it impractical to attempt to perform two complete tests in succession upon the same patient because of the progressive discomfort of the mouthpiece and noseclip and the drying of the mouth which results from spirometer breathing. A series of 15 patients was selected, therefore, whose disease was stable enough so that changes in pulmonary function would not be

expected to occur over short intervals. Retesting of these patients, at an interval of a few days to several weeks after the initial test, revealed very good agreement between the values of the two tests as shown in Table II. As illustrated by the last case in this series, and as we have observed not infrequently in subsequent experience with the test, patients with extremely prolonged resaturation times sometimes exhibit considerable quantitative variation in the actual resaturation time values, but in no case have such variations obscured the presence of a marked deficiency in each test.

Critique: Technical.—The outstanding feature of this test is its simplicity. A technician acquainted with basal metabolism testing can learn how to perform this test in a single day. The entire test, from the time the patient enters the

TABLE II. RESULTS OF RETESTS UPON THE SAME PATIENT

PATIENT	DATE	BASAL SATURATION (PER CENT)	RESATURATION TIME (SECONDS)
J. R.	7/28/49	96.8	11
	7/29/49	96.2	11
R. L.	10/25/49	96.7	12
	11/ 1/49	96.4	11
S. C.	2/10/50	96.9	11
	2/20/50	96.5	12
W. W.	3/28/50	97.2	15
	4/19/50	97.0	18
	4/26/50	96.8	15
T. H.	8/23/49	96.9	17
	10/12/49	97.0	16
R. F.	4/ 5/50	97.1	16
	4/18/50	97.1	20
C. B.	1/26/50	96.5	19
	2/14/50	96.5	21
P. F.	6/16/49	95.2	23
	8/23/49	96.1	22
R. S.	11/16/49	97.1	22
	1/25/50	96.4	27
R. J.	2/ 2/50	96.0	26
	3/ 7/50	96.5	23
G. M.	2/16/50	96.0	38
	2/27/50	96.2	27
R. C.	1/12/50	96.9	59
	3/15/50	96.8	50
D. K.	3/13/50	95.3	62
	3/23/50	94.9	55
H. G.	2/10/50	96.4	110
	2/12/50	96.9	101
L. W.	11/15/49	95.3	158
	1/30/50	94.8	69

laboratory until the final report is filled out, requires about 30 minutes. From the standpoint of the patient, it entails little more than the standard basal metabolic rate test which it closely resembles. Subjective symptoms of hypoxia are minimal. In contrast to many tests of pulmonary gas exchange which require breathing according to specific instructions, this test involves only natural respiration. It therefore is not dependent upon any significant degree of patient cooperation or patient intelligence. Small leaks around the mouthpiece or face mask, which constitute a major hazard in many pulmonary function tests, are of no importance provided that the leaks are not so great as to prevent attainment of the 80 per cent saturation level. This test may be carried out at the bedside of the patient who is acutely ill as well as on the ambulatory patient. Indeed we have encountered many instances where acute complications such as hemoptysis and severe chest pain have contraindicated the usual ventilatory tests, and oximetry appeared to be the only functional test that could be performed with safety.

A particular feature of safety in this test is that the degree of reduction in the oxygen tension in the inspired air is automatically determined by the patient's own functional capacities. Analyses of the oxygen content in the spirometer at the 80 per cent saturation level have revealed that, in the patient with severe functional impairment, this level may be reached at 13 to 15 per cent oxygen; while in others the inspired oxygen may fall to as low as 7 per cent before the 80 per cent saturation level is reached. One thereby avoids the possibility of precipitating an anginal attack in a patient with severe cardiopulmonary disease who is exposed to a predetermined level of restriction in the inspired oxygen tension.[14] It is for this latter reason that we terminate the hypoxia at the entirely safe level of 80 per cent saturation.

A source of variation in the resaturation times observed in normal subjects (Fig. 1) resides in the variable degree of respiratory stimulation associated with mild hypoxia in different subjects.[15, 16] Since the initial portion of the resaturation process is strongly influenced by the exact depth and rate of the first few breaths after the switch-over to air breathing, the pattern of the initial rise in the oximeter reading is quite variable. In addition, most individuals exhibit a posthypoxic depression in saturation, such that, after the return to room air breathing, the oximeter will rise to only 94 or 95 per cent and then often fall a point or two before slowly climbing to the original basal saturation level. The 80 to 90 per cent interval was therefore selected as the optimal index to resaturation, since it reduces the importance of the initial portion of the process but still stays comfortably below the posthypoxic depression in saturation. On this same basis, however, it is important to recheck the 100 per cent saturation setting on the oximeter when delayed resaturation times are observed. Errors of greater than 1.5 per cent in the oximeter reading could result in a confusion of the normal posthypoxic depression in saturation with an apparent prolongation of the 80 to 90 per cent resaturation time.

In spite of its variability, the respiratory stimulation during the hypoxic exposure is usually not more than 50 per cent above the basal ventilation rate. In occasional subjects, however, significantly greater degrees of hyperpnea are

observed which can introduce complications into the resaturation time determination. When such individuals are returned to air breathing, some will demonstrate a prompt acapneic depression of respiration which will tend to prolong the resaturation time. Others carry the hyperpnea over through the resaturation period and thereby abbreviate the resaturation time. In our experience, errors from this source do not become significant until hyperpnea in excess of 20 liters per minute is encountered. We therefore have adopted the practice of calculating the ventilatory rate at the end of the hypoxic period from the spirometer record. If the minute volume of respiration at this time is over 20 liters, the test is considered invalid and repeated. It should be understood that retesting for this reason is required in only a very small percentage of patients; nevertheless, in evaluating individual patients, a serious error might be made if complications of this nature were not recognized.

Critique: Theoretical.—When used for the routine evaluation of patients, it is obvious that this test in itself has limited value until it is correlated with the over-all clinical and laboratory findings. Like most tests of this nature, it does not indicate whether pulmonary deficiencies are due to primary pulmonary disease or to pulmonary disturbances secondary to cardiovascular disease. Its interpretation is also conditioned by the assumption that there are no abnormal vascular shunts by-passing the pulmonary bed and no abnormalities in the blood hemoglobin to invalidate the oximeter method.

The ability of this test to distinguish whether deficiencies are dominantly due to simple ventilatory defects or to diffusion defects cannot be compared in accuracy to the information that can be obtained by direct analysis of alveolar and blood gases.[3] On the other hand, it must be recognized that in many chronic lung diseases one would expect to find both types of deficiency present. Therefore the large proportion of patients with tuberculosis who showed mixed oximetric deficiencies (see Table I) is not unreasonable. From a clinical standpoint, it is the more extreme examples of the two types of deficiency which it is most important to identify, and it is felt that the test described here offers some real assistance in making this type of differentiation.

Originally it had been hoped that the interpretation could be carried somewhat further by obtaining more data upon respiration at the hypoxic level. Theoretically it should be possible to calibrate the system so that the amount of oxygen consumed by the patient, as indicated by the spirometer record, could be converted directly into the tension of oxygen in the inspired air at that time. This, correlated with the ventilation rate and saturation, should throw further light upon pulmonary gas exchange. Actual gas analyses together with pneumographic studies of changes in chest volume, however, revealed that changes in the tonus of inspiratory muscles associated with hypoxia[17, 18] so distort the volume of the lung-spirometer system as to invalidate such an analysis of the recordings.

The exact degree of significance which should be attributed to the mild deficiencies detected by this test is not always clear. In considering patients for pulmonary resection, for example, it is encouraging to find oximetry readings that are within normal limits. Such findings are not at all uncommon in

those cases where the diseased area has been essentially excluded from contributing to pulmonary function and where secondary processes have not resulted in diffuse damage to the remaining lung. On the other hand, in those cases where deficiencies in the oximetry readings are observed, there is as yet no adequate basis for assessing how severe a deficiency should be present in order to contraindicate major chest surgery. In our own experience, however, we have found this oximeter test, when weighed together with the over-all clinical picture, of considerable practical value, as may be best illustrated by the brief case reports given here.

ILLUSTRATIVE CASES

CASE 1.—R. K., a 43-year-old white man, had known pulmonary tuberculosis over a period of eight years with bilateral apical cavitation. Pneumoperitoneum, right phrenic paralysis, streptomycin, and PAS had served to control the disease considerably and closed the cavity on the right. The cavity in the left upper lobe persisted, and the patient was considered for major surgery.

Clinically this patient appeared to be only a fair operative risk. His appetite was poor and he was underweight. Chest roentgenogram revealed, in addition to the cavity on the left and dense scarring in the right apex, considerable scattered mottling throughout both lung fields with numerous stringy shadows.

In the laboratory he was found to have a vital capacity of 3.1 liters or 70 per cent of normal. Maximum breathing capacity was 61 liters per minute. The respiratory pattern showed definite abnormalities interpreted as loss of normal lung elasticity. Oximetry revealed a basal saturation of 96.8 per cent and a resaturation time of 90 seconds, indicating a severe deficiency of the diffusion type. On this basis the clinical impression of a fair operative risk was revised to that of a poor operative risk and major surgery was deferred.

This case illustrates a common problem in chronic pulmonary tuberculosis. Deficient pulmonary function must always be suspected in the presence of radiological evidence of diffuse fibrosis, but we have observed many such cases in which pulmonary function remained reasonably good. Knowledge of the functional significance of these fibrotic shadows, as obtained by the oximeter test, may aid considerably in formulating the proper course of treatment.

CASE 2.—R. H., a 21-year-old white man, had a right lower lobectomy two years previously for bronchiectasis. Pathologic examination of the specimen revealed the surprising findings of caseation necrosis and acid-fast bacilli. On this basis the right hemidiaphragm was paralyzed and the patient placed on pneumoperitoneum therapy and bed rest. After a few months on this regime, the patient left the hospital against medical advice and continued the pneumoperitoneum under the care of a private physician. One year later he was admitted to Brecksville Hospital with hemoptysis. Examination revealed bilateral spread of the disease, with dense pneumonic disease and cavitation throughout the right upper and middle lobes and scattered infiltrations throughout the left lung. A course of streptomycin therapy controlled the disease on the left and led to some resolution on the right, although dense infiltration with probable cavitation persisted. The patient was in fair general condition, but complained of some shortness of breath on mild exertion.

Laboratory examination revealed a vital capacity of 1.9 liters or 46 per cent of normal and a maximum breathing capacity of 69 liters per minute. Oximetry yielded a basal saturation of 95.6 per cent and a resaturation time of 16 seconds. The slightly low basal saturation was attributed to a ventilatory defect associated with the pneumoperitoneum. The normal resaturation time was taken as evidence that there was no significant physiologic damage in the functioning left lung.

It was elected to perform a thoracoplasty on the right. The postoperative course was uneventful. Three months postoperatively the ventilatory capacity showed a significant increase, associated with the greater expansion of the left lung due to discontinuance of the pneumoperitoneum. The basal saturation had risen to 96.9 per cent and the resaturation time was 11 seconds.

This case represented a difficult clinical decision. Some would favor radical resection of the remaining right lung. Others, in view of the past history including recent disease scattered throughout the left lung, would hesitate to embark upon major surgery. Knowledge of the functional status of the left lung, as obtained by the oximeter, can aid in making the decision.

CASE 3.—H. M., a 51-year-old white man, had a left pneumonectomy two years previously for carcinoma. Since then the patient had complained of severe dyspnea. His local physician had informed him that he suffered from compensatory emphysema of the remaining lung and that there was nothing that could be done for the condition. On this admission the patient was markedly dyspneic, orthopneic, and had become a respiratory cripple. Cyanosis was absent. Cardiac findings were negative and there was no evidence of a recurrence of malignancy. X-ray examination of the chest showed some mediastinal shift and distention of the right lung without definite emphysema.

Laboratory study revealed a vital capacity of 2.0 liters or 64 per cent of normal and a maximum breathing capacity of 70.6 liters per minute. Oximetry gave a basal saturation of 95.2 per cent and a resaturation time of 16 seconds. It might be noted that at the start of this test the patient's attention was called to the fact that "the machine was filled with pure oxygen" and no dyspnea was observed during the oximeter test.

It is clear that the remaining right lung in this patient had undergone some degree of distention, but the finding of a resaturation time that was well within normal limits is considered to exclude any significant degree of functional emphysema. The dyspnea must therefore be explained in terms of a respiratory neurosis. This possibility had been entertained clinically, but, with a known etiological factor for emphysema present, it would have been difficult to deny a pulmonary basis for the crippling respiratory distress without the evidence of the oximeter.

It might be noted that we have encountered a number of similar cases in which the oximeter test has been invaluable in establishing the presence or absence of a pulmonary basis for a complaint of dyspnea. Unlike ventilatory tests, these oximeter readings cannot be distorted by lack of effort, willful or otherwise, on the part of the patient.

SUMMARY

1. A test is described for evaluating the efficiency of pulmonary gas exchange, employing the earpiece oximeter and a basal metabolism machine. After obtaining the basal saturation of the patient while breathing room air, his saturation is lowered to 80 per cent by having him rebreathe into the air-filled spirometer. The patient is then quickly switched to room air breathing and the time is measured for the saturation to rise from 80 to 90 per cent.

2. Measurements on 55 healthy adults indicate a normal resaturation time as determined by this test of 13.4 ± 3.4 seconds.

3. Results of this test upon 300 patients with pulmonary tuberculosis but without clinical cyanosis are reported. Deficiencies were demonstrated by this

test in 53 per cent of these patients. In 38 per cent of the patients exhibiting deficiencies, it was possible to estimate whether the deficiency was dominantly due to reduced ventilation or to impaired gas diffusion.

4. Retesting the same patients revealed adequate reproducibility of the test values for the routine use of the test in patient evaluation.

5. Technical and theoretical limitations of the method are discussed. The clinical use of the method is illustrated with case histories.

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SUCCESSFUL CONSTRUCTION OF AN ARTIFICIAL ANTETHORACIC ESOPHAGUS

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IT IS THE purpose of this paper to report further on a method of replacement for an esophagus, originally reported by Longmire and Ravitch,[1] which will occasionally be found useful. Reconstruction was necessary because of a severe stricture of the esophagus complicated by perforation, mediastinal abscess, esophageal-mediastinal-bronchial fistula, and chemical burns of the upper portion of the stomach.

The discomfort, inconvenience, and complications attendant upon gastrostomy and/or repeated esophagoscopy and bouginage make patients with a severely stenotic esophagus desirous of reconstruction. Prevailing methods use the stomach, jejunum, or tubes of skin. The stomach or the jejunum may be brought up either inside or outside the thorax.

If the distribution of the blood supply to the proximal jejunum is satisfactory, the method of Roux,[2] popularized by Yudin,[4] will be found best for routine use. Placing the stomach in the chest is not physiologic and is not well tolerated in older patients where gastric dilatation may produce uncomfortable fullness or induce episodes of cardiac irregularity. When the arrangement of the blood supply of the jejunum precludes the Roux[2] procedure and a previous injury prevents the intrathoracic transfer of the stomach, as in the patient to be reported, the operation described by Longmire and Ravitch[1] will be found useful. Based on experiments in dogs by Stafford,[3] the technique consists of transplantation and migration of a segment of jejunum in the subcutaneous tissues to effect a new union between the esophagus and stomach. The method is well described and beautifully illustrated in the paper of Longmire and Ravitch.[1]

CASE REPORT

L. W. (B-26,269), a 25-year-old Negro man, was admitted to the medical service of the Veterans Administration Hospital, Fort Howard, Md., with a history of swallowing a corrosive liquid four days previously. A Levin tube was easily passed into the stomach for feeding, and removed eight days later when the patient swallowed with minimal discomfort. At this time, demonstration of an esophageal-bronchial fistula and a mediastinal abscess by roentgenogram after the administration of Lipiodol (Figs. 1 and 2) made gastrostomy necessary. The fundus of the stomach was found to be severely burned so the gastrostomy was done near the pylorus. Later, the mediastinal abscess was drained extrapleurally after resection of about 5 cm. segments of the right eighth, ninth, and tenth ribs. The fistula was found at the level of the azygos vein. The opening in the esophagus was sutured and the closure reenforced by fascia from the latissimus dorsi muscle. The wound, which was left open, healed uneventfully.

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Fig. 1.



Fig. 2.



Fig. 3.

Fig. 1.—Demonstration of mediastinal abscess and bronchogram of lower lobe of right lung.
Fig. 2.—Demonstration of fistula between esophagus to mediastinum to lung.
Fig. 3.—Roentgenogram of antethoracic replacement after administration of barium.

Damage to the esophagus was severe, as demonstrated by the progressive stricture which ultimately extended to a point about 1½ cm. above the clavicle. Attempts to pass a piece of black silk through the esophagus were fruitless. Performance of a minor plastic procedure on the gastrostomy afforded an opportunity for retrograde bouginage but an obstruction was encountered at the esophagogastric junction.

Since some type of reconstruction was desirable, the conditions present made an antethoracic replacement using a jejunal graft the procedure of choice. At laparotomy, a segment of jejunum about 30 cm. in length, with two centrally located arteries and veins, was isolated, starting about 16 cm. below the ligament of Treitz and placed outside the abdomen. Intestinal continuity was restored by end-to-end anastomosis. A later complicating episode of acute intestinal obstruction was relieved by operative division of adhesions constricting the jejunum about 30 cm. below the anastomosis. A series of several operative procedures was necessary in order to cover the loop of jejunum with skin, divide the central pedicle, delay each end of the graft to insure an adequate blood supply, transfer the graft, close the



Fig. 4.—Patient about one and one-half years after discharge.

gastrostomy, and restore continuity of the alimentary tract. Despite creation of an apparently adequate anastomosis between esophagus and jejunum, a stricture developed and was corrected surgically. The patient was now able to eat a normal diet and was discharged from the hospital.

Approximately one month later, readmission for treatment of stricture again at the proximal anastomosis was necessary. A catheter for feeding purposes was inserted through an opening in the upper part of the loop of jejunum and a minor revision of the anastomosis was done subsequently.

Stricture formation, however, again returned necessitating further revision. The anastomosis and surrounding scar tissue were excised. Resection of the medial 3 cm. of the clavicle facilitated the making of a large end-to-end anastomosis. The postoperative course was uneventful. The patient has remained well for about one and one-half years. (Figs. 3 and 4.)

COMMENT

The procedure provides an isoperistaltic tube lined with mucous membrane as a replacement for the esophagus. A more physiologic passage was provided than by the Roux procedure since the stomach is not by-passed and the risk of formation of a gastric or duodenal ulcer is, therefore, seemingly reduced. A jejunal ulcer from regurgitation of acid gastric contents might result but the isoperistaltic movements of the jejunum should minimize this hazard.

The major objection to the procedure is the time required for performance which limits its use to benign diseases of the esophagus. Patience is necessary since stenosis of the anastomosis and fistulas are troublesome although solvable problems.

SUMMARY

The method of Longmire and Ravitch[1] using a free jejunal graft was successfully employed in the construction of an artificial antethoracic esophagus. Esophageal-mediastinal-bronchial fistula, mediastinal abscess, chemical burn of the stomach, and a blood supply to the proximal part of the jejunum precluding transposition were features making this type of reconstruction advisable.

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THE PATENT DUCTUS

A REVIEW OF SEVENTY-FIVE CASES WITH SURGICAL TREATMENT INCLUDING AN ANEURYSM OF THE DUCTUS AND ONE OF THE PULMONARY ARTERY

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IT MAY be categorically stated that a ductus arteriosus persistently patent after the fifth year of life, with or without symptoms, should be closed to avoid certain potential hazards. These are retardation in physical and perhaps mental development, cardiac dilatation and decompensation, and bacterial endocarditis and endarteritis. Should any of these complications appear before the age of 5, immediate operative closure of the ductus is indicated.[1] One infant in our series was operated upon for cardiac failure at the age of 16 months, and we have just operated upon a second infant 5 months old in incipient cardiac failure with a pulse rate of 160 to 180, a respiratory rate of 80 to 100, and a blood pressure of 110/0. Another lesion may well be present but definite improvement has occurred as shown by a present blood pressure of 100/68. On the other hand, Josefson[2] reported a patient who reached the age of 66 years before any harmful effects of the open ductus appeared. A woman of 79 died recently at San Francisco Hospital who was known to have had the murmur of an open ductus for many years, with cardiac symptoms for only about five years. At autopsy, a "small" patent ductus with great dilatation of the pulmonary artery and generalized cardiac hypertrophy were found. Why should there be this absence or long delay in the development of symptoms? Why in Maude Abbott's series[3] of ninety-two patients with patent ductus should 43 per cent die of cardiac failure at an average age of 24 years? In another series,[4] 71 per cent of the patients died before the age of 40, largely of cardiac complications. Writers generally speak in vague terms of "an increased burden on the heart as the patient grows older, which manifests itself by cardiac enlargement." Others emphasize only the size of the ductus as the determining

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factor in the development of cardiac symptoms. A sounder and more accurate explanation may be found in the abnormal physiology of the patent ductus, which is comparable in many respects with the pathologic physiology of a peripheral arteriovenous fistula.[5] The ultimate effect of a large arteriovenous fistula is cardiac dilatation, decompensation, and death if not closed by operation. These effects are dependent upon the deflection of a large volume of blood from the arterial bed with its high pressure directly into the capacious venous bed with its low or even negative pressure.[6, 7] Because of the low or absent resistance at the site of the fistula as compared with the capillary bed elsewhere, blood flows more freely through the fistula into the venous bed and back to the heart, thus increasing the cardiac output by the volume of blood flowing through the fistula and, in effect, engrafting upon the normal circulation a shorter parasitic circulation.[8]

The volume of blood deflected through the fistula is controlled by the size of the fistula, by its distensibility, and by distensibility of the vessels leading to and from the fistula. Given distensible vessel walls with compressible tissues in which these vessels lie, and an absence of fibrosis at the site of the fistula, the volume of blood flowing toward and through the fistula with its low resistance will tend always to increase. The result is a progressive dilatation of that part of the circulatory bed through which the short-circuited blood flows, including the artery and vein leading to and from the fistula, both sides of the heart, and the fistula itself. Recorded cases of peripheral fistulas indicate that the size of the fistula does not remain stationary, but may increase from 5 to 20 mm. in diameter over a period of 20 to 25 years. As the fistula dilates and the volume of short-circuited blood increases, all chambers of the heart become dilated, and decompensation is imminent and inevitable if the fistula is not closed. In the growing animal,[9] experimentally produced peripheral fistulas have resulted in remarkably large hearts characterized both by great dilatation and by great hypertrophy. It was suggested that when dilatation outstrips hypertrophy, as might be expected in the adult animal, decompensation is an early sequence, but when increasing hypertrophy parallels and is commensurate with the increasing dilatation, as might occur in a growing animal, great cardiac enlargement is possible without decompensation.

A patent ductus produces comparable effects upon the circulation with certain unique modifications: in intrauterine life, the right heart pumps blood through the ductus from the pulmonary artery to aorta against the peripheral resistance of the systemic circulation. In the fetus the circulating blood is about equally divided in volume flow between the right and left hearts. Up to birth, therefore, the two ventricles have been propelling forward equal volumes of blood against equal peripheral resistances with the result that normally at birth the two ventricles are equally developed, and both contribute equally to the formation of the apex of the heart.[6] Immediately after birth if, for some still unknown reason, the ductus remains open, the large reservoir of low resistance in the pulmonary bed attracts blood to it from the systemic circulation and causes a reversal of flow through the ductus which then trans-

ports blood from aorta to pulmonary artery (Fig. 1). It is apparent at once that under these circumstances the blood flowing through the ductus will flow only through the pulmonary artery, the lungs, the pulmonary veins, the left heart, and the aorta, completely by-passing the systemic circulation, the vena cava, and the right heart. The normal cardiac output of the left ventricle is thus greatly augmented by the volume of blood flowing through the ductus

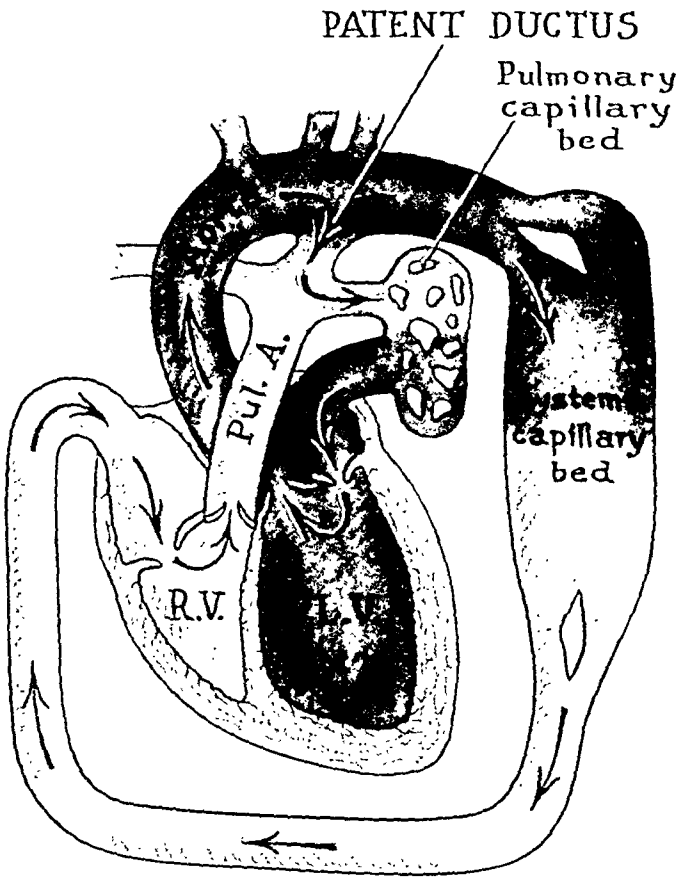


Fig. 1.—Schema of circulation in presence of a patent ductus transporting blood from aorta to pulmonary artery, resulting in dilatation of the left heart. The low resistance in the pulmonary arterial bed will continue to attract an increasing volume of blood through the ductus, resulting in its dilatation and in a progressive impairment of the circulation.

back to the heart. This short-circuited blood serves no purpose and may be considered parasitic upon the normal circulation. Eppinger and Burwell[10] demonstrated in a patient with a ductus that 25 liters of blood per minute flowed through the ductus and the left heart, and only 5 liters per minute flowed through the right heart. Closure of the ductus equalized the flow through the two ventricles to a little over 5 liters. The inevitable consequence of this greater volume flow of blood through the shorter circuit is a dilated left heart. The right heart, through which the smaller or normal volume of

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ing normally after birth remain easily distensible, a characteristic which may persist throughout life, permanently preventing its automatic closure once it has provided a pathway to the reservoir of low resistance in the pulmonary bed. Indeed, failure to close in the first instance may be a direct result of the very low resistance in the pulmonary bed which attracts blood to it through the ductus. A flow once established would be an effective factor in maintaining the patency of the ductus, particularly if the differential in the resistances of the two systems is high. With the passage of time the difference in these resistances, though significant and effective, may lessen due to this factor of increasing fibrosis, and the tendency for the volume flow of blood through the ductus to increase is, therefore, correspondingly lessened. Hence, the disabling effects of the ductus and of the parasitic circulation which it produces, are slow in developing, and may be delayed for years, but they are nonetheless inevitable if the ductus remains patent, if its walls remain easily distensible, and if progressive dilatation of the ductus is not prevented by its inherent fibrosis or by the fibrosis of inflammation and infection. Just as a peripheral fistula, if large, may produce cardiac failure in three weeks, or if small may remain comparatively symptomless for many years and then end in cardiac decompensation because of progressive dilatation of the fistula, so may the harmful consequences of a patent ductus appear soon after birth or be delayed for many years. The actual course of events is determined by the degree of difference of resistances in the pulmonary and systemic beds, which in turn determines the degree of difference in the aortic and pulmonic pressures, and by the size, distensibility, and progressive dilatation of the ductus itself.

Certain authors contend that surgical ligation is indicated for the uncompensated patent ductus at whatever age signs of circulatory embarrassment appear, but for patients with a "compensated" ductus a policy of watchful waiting is advised. Our view is that since the open ductus provides a permanent passageway from a bed of high resistance to a bed of low resistance, it will never be fully "compensated," and will always tend to transport more and more blood within the limits of its distensibility or until the resistances in the two beds are more or less equalized. It is this concept of the pathologic physiology of a patent ductus that justifies the categorical statement that a ductus must be closed, preferably early in childhood, before the hazards of impairment of physical growth and cardiac decompensation arise.

The peripheral fistula and the patent ductus are also quite comparable in their effect upon blood volume and blood pressure.[11] With the opening of an experimentally produced peripheral fistula, there occurs a prompt fall in blood pressure which is compensated for by an increase in total blood volume commensurate with the fall in pressure and with the volume of short-circuited blood. If the fistula is small, the systolic and diastolic pressures may be restored to normal by this increase in blood volume. If the fistula is large, the diastolic pressure is permanently lowered due to the significant decrease in peripheral resistance at the fistula. When a peripheral fistula is closed by operation, there is a reversal of these changes (Fig. 3, *A*)—the systolic and

blood flows, remains small. Indeed, the volume of blood flowing through the systemic circulation may be so reduced as to interfere with proper mental and physical growth. A number of our young patients blossomed in vigor and growth promptly after closure of the ductus, and one of two children (Fig. 2) who showed mental retardation in addition to other defects was apparently improved after elimination of the ductus. The literature contains other reports of the association of patent ductus with mental inadequacy.[5]

These pathologic effects of the abnormally patent ductus depend upon the low resistance to the flow of blood encountered in the pulmonary circulation, as compared with the high resistance to the flow of blood through the systemic capillary bed. Since a difference in the resistances of the two systems usually persists throughout life, and since, in consequence, the aortic pressure is



Fig. 2.—Two children with unilateral cataract, strabismus, some mental impairment, and patent ductus. (Each mother had rubella at one month of gestation). Ligation of ductus has resulted in definite improvement in mental status of child *a* and apparent improvement in child *b*.

permanently greater than pulmonic pressure, the tendency will be for blood to be deflected in increasing volume to the bed of less resistance. The effect of this increasing volume flow is progressive dilatation of that part of the circulatory bed through which the blood flows, including the ductus, and the left heart. Certain factors may limit or even prevent this increasing volume flow through the ductus, in which case the effects of the ductus will remain stationary throughout life. These factors are the resistance to dilatation inherent in the fibrous wall of the ductus itself, and the increasing fibrosis in the pulmonary capillary bed subjected for a prolonged period to the high arterial pressure of the systemic circulation.

The failure of a ductus to close completely may perhaps be due to a deficiency in fibrosis in the structure of its walls which, instead of contract-

ing normally after birth remain easily distensible, a characteristic which may persist throughout life, permanently preventing its automatic closure once it has provided a pathway to the reservoir of low resistance in the pulmonary bed. Indeed, failure to close in the first instance may be a direct result of the very low resistance in the pulmonary bed which attracts blood to it through the ductus. A flow once established would be an effective factor in maintaining the patency of the ductus, particularly if the differential in the resistances of the two systems is high. With the passage of time the difference in these resistances, though significant and effective, may lessen due to this factor of increasing fibrosis, and the tendency for the volume flow of blood through the ductus to increase is, therefore, correspondingly lessened. Hence, the disabling effects of the ductus and of the parasitic circulation which it produces, are slow in developing, and may be delayed for years, but they are nonetheless inevitable if the ductus remains patent, if its walls remain easily distensible, and if progressive dilatation of the ductus is not prevented by its inherent fibrosis or by the fibrosis of inflammation and infection. Just as a peripheral fistula, if large, may produce cardiac failure in three weeks, or if small may remain comparatively symptomless for many years and then end in cardiac decompensation because of progressive dilatation of the fistula, so may the harmful consequences of a patent ductus appear soon after birth or be delayed for many years. The actual course of events is determined by the degree of difference of resistances in the pulmonary and systemic beds, which in turn determines the degree of difference in the aortic and pulmonic pressures, and by the size, distensibility, and progressive dilatation of the ductus itself.

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diastolic pressures are temporarily elevated but fall again as the previously increased blood volume returns to normal. The elimination of a peripheral fistula in each of three patients caused a reduction in total blood volume of 2,700, 1,000, and 1,200 c.c., respectively,[12] If a fistula is large, its closure will produce a permanent elevation of diastolic pressure due to the significant increase in peripheral resistance.

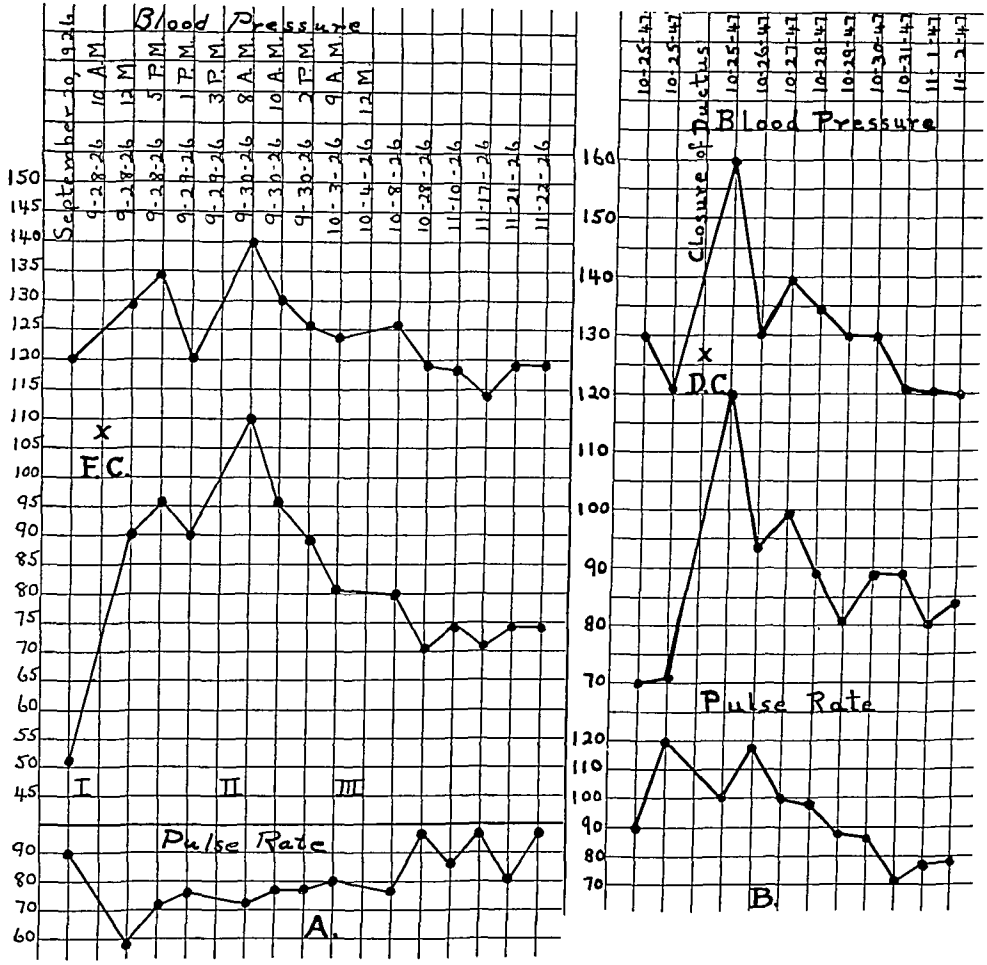


Fig. 3.—Similarity of blood pressure response to closure of fistula (Chart *A*), and closure of ductus (Chart *B*): The temporary abnormally high elevation of diastolic and systolic levels is due to temporary overdistention of the circulatory bed by the increase in total blood volume, which accompanies both these lesions. Diastolic pressure is permanently elevated due to increase in peripheral resistance.

Similarly, the production of a patent ductus by anastomosis of the left subclavian artery to the left pulmonary artery, as in the Blalock procedure, causes a fall in blood pressure, chiefly diastolic. That the blood volume is increased in the presence of a ductus was demonstrated by Eppinger, Burwell, and Gross,[13] who showed a reduction in blood volume following closure of a ductus in one case from 3,980 to 3,280 c.c., and in a second case from 4,130 to 3,720 c.c.

Closure of a patent ductus by operation is accompanied by a temporary abnormal increase both in systolic and diastolic pressures (Fig. 3, *B*), which then drop to lower levels as the total blood volume falls to normal. If the ductus is large, the diastolic pressure remains permanently elevated, due again to a significant increase in peripheral resistance.

The following case report illustrates how very early the symptoms of cardiac disability may occur in the presence of a patent ductus.

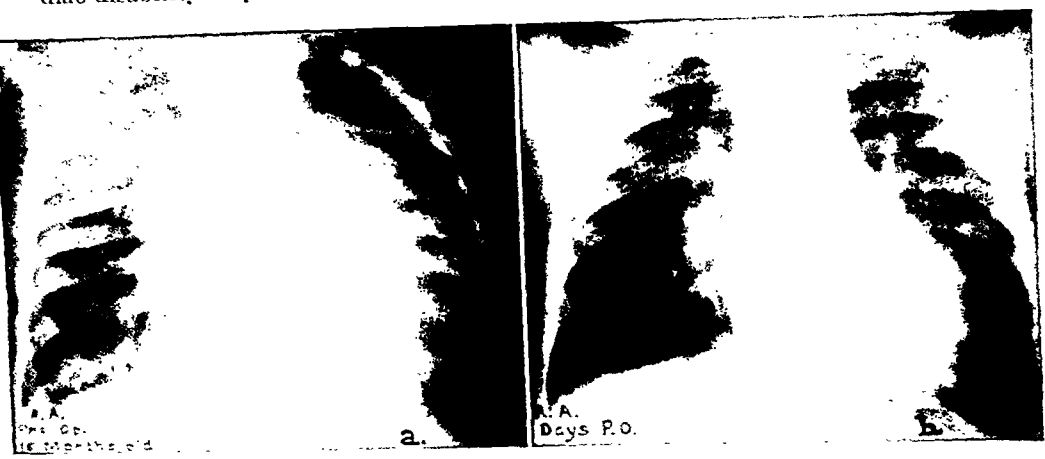


Fig. 4 (Case 1).—*a*, At 16 months of age the patient was in incipient cardiac failure due to patent ductus with marked dilatation of left heart. *b*, Twenty-six days after closure of the ductus, marked reduction in size of left heart had already occurred.

CASE REPORTS

CASE 1.—R. A., 16 months old, had had a heart murmur since he was 2 weeks of age. This was accompanied by attacks of dyspnea and a cough ascribed to recurrent pulmonary infection, but for which oxygen administration was necessary on several occasions. A roentgenogram taken at the age of 3 months showed cardiac enlargement. Thereafter, other films showed a progressive increase in heart size and prominence of the pulmonary vascular markings. At 15 months it was necessary to digitalize the patient in an effort to control heart failure. The murmur at this time was continuous and was heard maximally in the left second interspace. The blood pressure was 136/0 in the right arm; the electrocardiogram was normal. A diagnosis of patent ducturs arteriosus was made, and on Oct. 10, 1951, a ductus measuring 8 by 8 mm. was obliterated. In twenty-six days the heart returned to normal size (Fig. 4). The child had no further dyspnea and developed rapidly. Except for an inguinal hernia, he is normal in all respects.

Another patient with a history of mild cyanosis since birth died in cardiac failure at the age of 16 years without operation, a clinical diagnosis of interauricular septal defect having been made. At necropsy, a patent ductus was found as the only lesion, with unmistakable evidence that the flow of blood was and always had been, as in intrauterine life, from pulmonary artery to aorta (Fig. 5). This example of the association of cyanosis with patent ductus as the only anomaly is important since it is generally taught that it never occurs except in combination with other anomalies.[1, 21]

CASE 2.—J. J., aged 16 years was admitted to Stanford Hospital in acute cardiac failure of about five days' duration. At 9 months of age, following an attack of whooping cough,

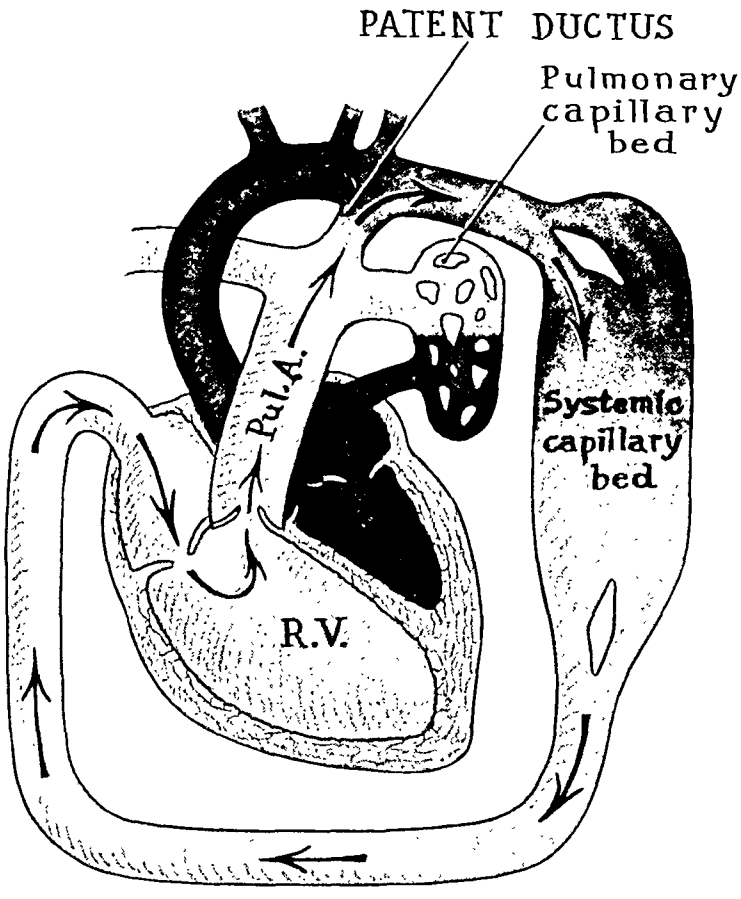


Fig. 5 (Case 2).—Schema of circulation when blood flows through ductus from pulmonary artery to aorta. The greater volume of blood flows through the right heart causing its dilatation. Hypertrophy also occurs because it is laboring against systemic resistance.

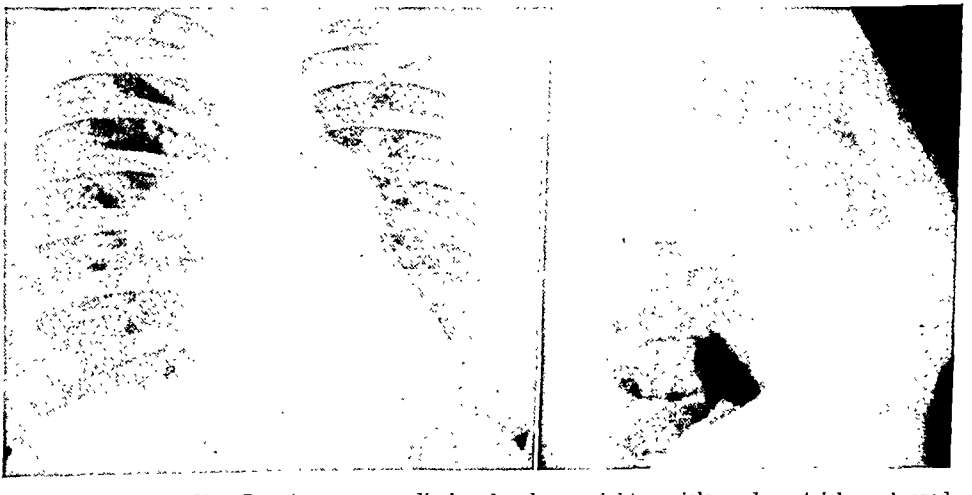


Fig. 6 (Case 2).—Roentgenograms disclosed a large right auricle and ventricle, enlarged pulmonary artery, and small left auricle and ventricle in presence of ductus transporting blood from pulmonary artery to aorta.

a cardiac murmur had been discovered. In recent years activity had become increasingly limited by fatigue and dyspnea. Although always a little flushed about the lips, she had been truly cyanotic only in the six months preceding admission, accompanied by increasing dyspnea and fatigue on exertion. A week before admission, vomiting occurred, which continued until she was placed in an oxygen tent. On physical examination, the child was well nourished, a little plump, with a distinct bluish color of lips and nails. She was dyspneic lying in bed.

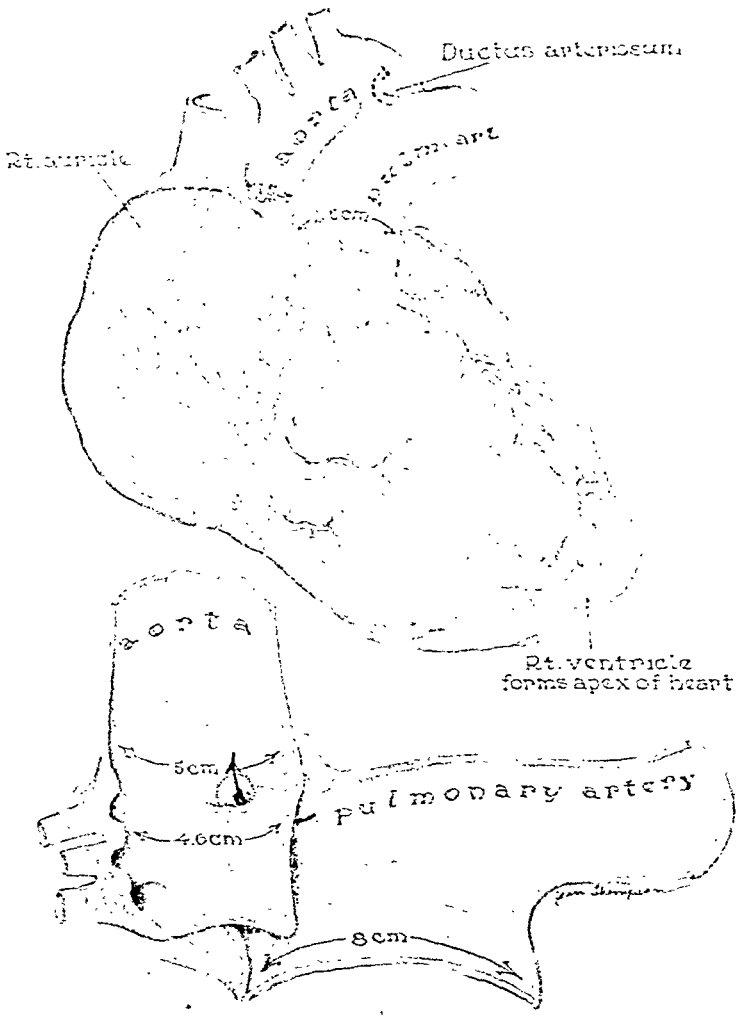


Fig. 7 (Case 2).—There is great dilatation of right auricle, and dilatation and hypertrophy of right ventricle which form the apex of the heart in presence of patent ductus transmitting blood from pulmonary artery to aorta. Note the excessive diameter and circumference of pulmonary artery as compared with aorta, and enlargement of aorta at site of entrance of ductus.

The chest and lungs were normal. The heart was slightly enlarged. There was a loud murmur maximal in the third interspace on the left, not well heard in the lung fields. It was thought to be systolic in timing. Blood pressure was 110/97, pulse rate 86.

Roentgenograms of the heart (Fig. 6) were interpreted as showing a very prominent and unusually large pulmonary artery, a bulging right auricle, a small left auricle, a small left ventricle, and small aorta. On fluoroscopy, a vigorous hilar dance was noted. The elec-

trocardiogram showed a marked right axis deviation, and abnormal P waves. The red cells numbered 6,100,000; hemoglobin 110 per cent or 18.7 Gm. per cent; the white cells numbered 8,450, 62 per cent polynuclears, 26 per cent lymphocytes, 8 per cent monocytes, 3 per cent basophiles. The urine had a specific gravity of 1.026 with 3 plus albumin and many pus cells. Cultures yielded *Escherichia Coli.*

Despite continuous oxygen administration the patient gradually became more cyanotic, dyspneic, and somnolent, and died thirteen days after admission. The clinical diagnosis was interauricular septal defect.

At necropsy, the heart weighed 400 grams. There was a remarkable hypertrophy of the right ventricle, and dilatation of the right auricle. The apex of the heart was formed by the right ventricle (Fig. 7). The left auricle and ventricle were small. The pulmonary artery was quite large, its circumference just beyond the valve being 8 cm.; at a corresponding point, the aorta was only 4.5 cm. in circumference. At the entrance of the patent ductus, the aorta measured 5 cm. in circumference. The tricuspid valve measured 11 cm. in circumference; the mitral valve 8 cm. in circumference. The right ventricle when dissected out weighed 167 Gm.; the left ventricle weighed only 78 Gm. Microscopically the fibers from the right ventricle were slightly larger than those of the left, and the nuclei were generally larger and more blunt.

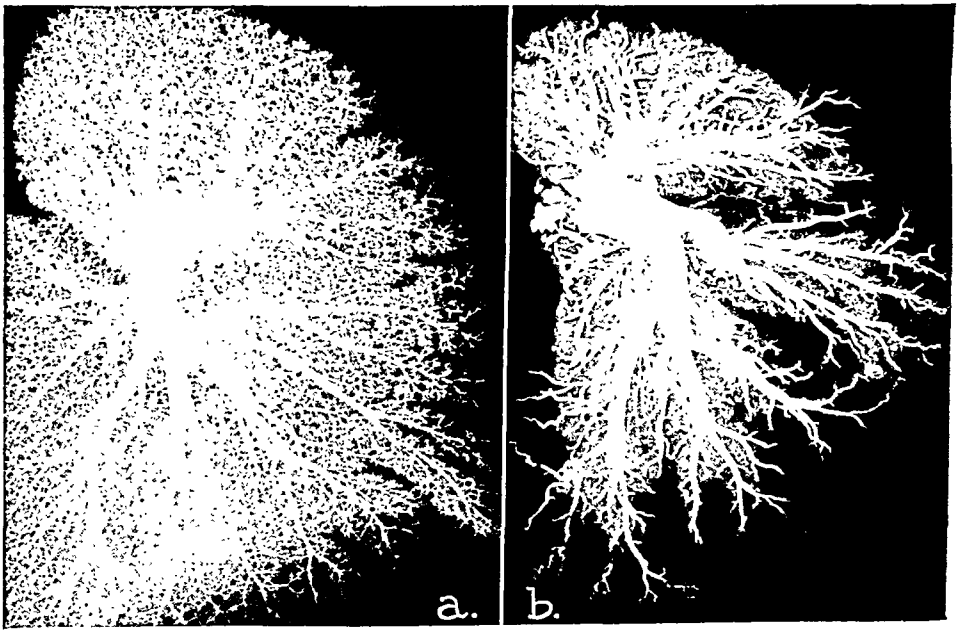


Fig. 8 (Case 2).—*a*, Roentgenogram of normal lung following injection with lead carbonate and mercuric sulfide in gelatin. *b*, Roentgenogram of lung (Case 2) similarly injected revealed evidence of pulmonary arteriosclerosis in the presence of a patent ductus through which blood flowed from pulmonary artery to aorta.

The pulmonary arterial tree was injected with lead carbonate, plus mercuric sulfide suspended in gelatin. When compared with a normal lung similarly injected (Fig. 8), it showed a truncated, blunted arterial tree, suggesting long-standing pulmonary hypertension and arteriosclerosis. Microscopically many of the small arteries and arterioles had thickened walls and narrowed lumina, and most of these had thickened intima with reduplication of the elastic laminas. A cross section of the aorta, patent ductus, and pulmonary artery showed the wall of the pulmonary artery definitely thicker than the aortic wall, and except for a definite heavy elastica interna, it was similar in structure.

A very exacting search revealed no lesion other than the patent ductus arteriosus which in the relaxed state measured 0.5 cm. in diameter but could easily be distended to 1 cm. in diameter. All evidence indicated that for her entire lifetime, this patient's ductus had been transporting blood from the pulmonary artery into the aorta,[4] thus providing a larger volume flow of blood through the right heart and systemic circulation than through the left heart and pulmonary circulation. The expected response to this increased volume flow was: (1) a preponderant dilatation of the right heart because of the larger volume of blood flowing through it; (2) a preponderant hypertrophy of the right heart due to its being in direct communication with the peripheral resistance of the systemic circulation against which it had labored for a lifetime; (3) a marked right axis deviation; (4) a pulmonary artery whose lumen was larger than that of the aorta with a wall thicker than the aortic wall; (5) an aortic lumen which was 4.5 cm. in circumference at its origin and 5 cm. at the entrance of the ductus; (6) a definite cyanosis in life, indicating an admixture of venous blood to the systemic blood by way of the ductus; (7) a high diastolic pressure since blood flowed through the ductus into the systemic circulation during diastole rather than out of it.

Summed up, these deviations provide unequivocal evidence that the flow of blood through the ductus was in this instance from pulmonary artery into the aorta. Seldom if ever has this type of ductus been recognized by the surgeon at the operating table, probably because this rarely encountered condition is usually mistakenly diagnosed, and surgery is not considered advisable. This may have been fortunate, as it seems highly probable that closure of the ductus in this 16-year-old girl with such a preponderantly developed right heart would have been incompatible with life. Recognition of this condition at an earlier age might permit successful surgical correction.

A second equally serious threat which may occur at an early age is a bacterial endocarditis and endarteritis involving the ductus itself, the adjacent pulmonary artery, or the aorta.[14] In the eighty cases of patent ductus studied by Bullock and his associates,[4] fatal bacterial endocarditis occurred in 53 per cent. Four of our cases were complicated by an endarteritis and a bacterial septicemia, one when the patient was 7, one at 10, and two at 11 years of age.

CASE 3.—I. R., a thin underdeveloped girl, was first seen at the age of 7, in November, 1944, for a cardiac murmur since birth. A diagnosis of patent ductus and megacolon was made. Operation on the ductus was not advised by the pediatricians because "the child had had no symptoms." Exactly one month later she was readmitted to the hospital acutely ill with high fever, marked anemia, and subacute bacterial endarteritis, for which she received over 12,000,000 units of penicillin in the course of three months' hospital care with subsidence of all symptoms.

She re-entered the hospital in January, 1946, perfectly well, but operation was now considered advisable as a prophylaxis against future trouble. Closure of a large ductus, 1 cm. in diameter, by multiple ligations was followed by a gain of fourteen pounds in a year's time, and by a marked reduction in the size of the heart (Fig. 9) comparable with the reduction in cardiac size that follows excision of a peripheral arteriovenous fistula. A blood pressure of 105/60 rose immediately after operation to 134/96. The following day a blood pressure of 138/110 was found, a week later 120/82, and two years later 115/80.

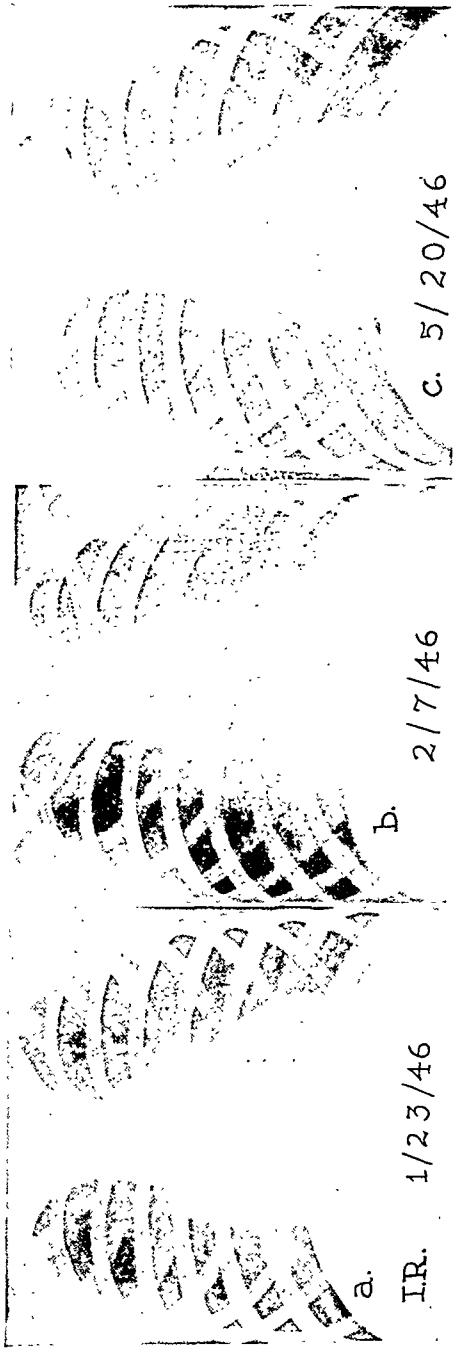


Fig. 9 (Case 3).—*a*, Large heart in presence of a patent ductus complicated by bacterial endarteritis. *b*, immediately following ligation of ductus. *c*, Three months later there is marked reduction in size of heart comparable with the reduction of cardiac size that follows elimination of a peripheral fistula.

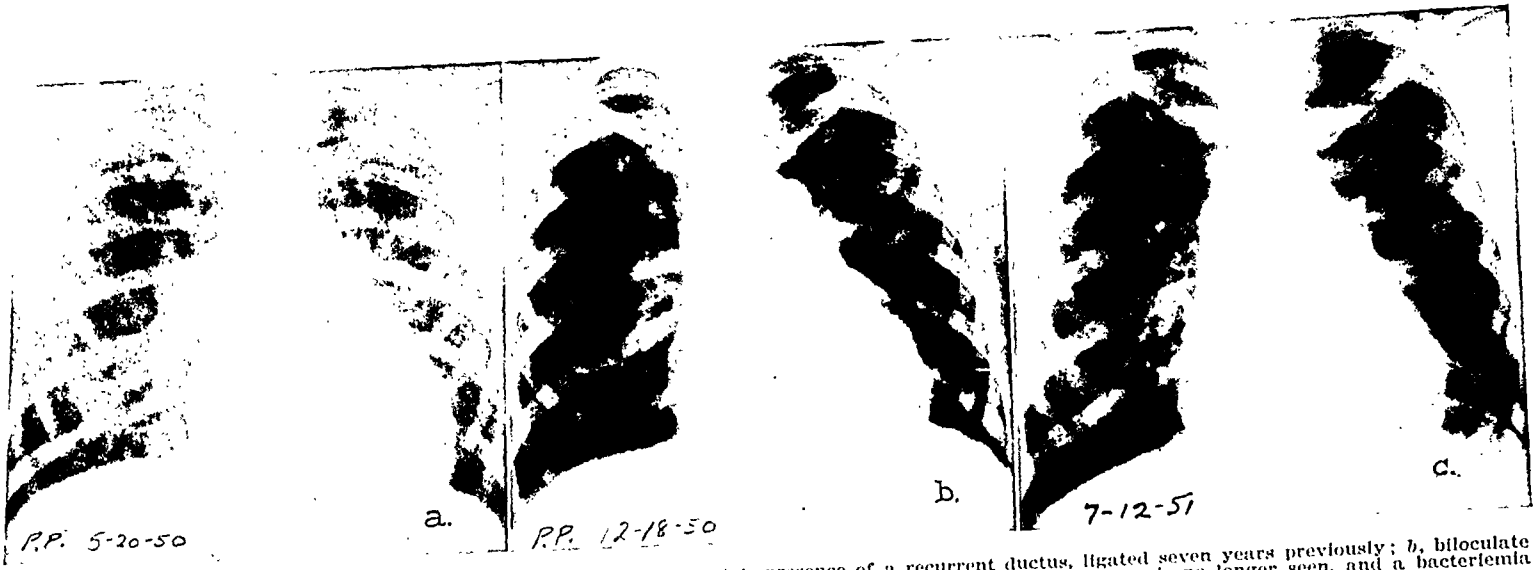


Fig. 10 (Case 4).—Roentgenograms disclosed *a*, dilatation of heart in presence of a recurrent ductus, ligated seven years previously; *b*, biloculate aneurysm of ductus at site of triple religation, ductus again patent; *c*, following division of ductus, the aneurysm is no longer seen, and a bacteriemia previously resistant to all antibiotic therapy was cured.

In two cases of open ductus complicated by bacterial endarteritis and septicemia, aneurysmal dilatations occurred, one of the ductus itself and one of the pulmonary artery.

CASE 4.—P. P., aged 11 years, was found to have a patent ductus at the age of 9 months. In 1943, at 4 years of age, because of shortness of breath and an enlarging heart, the ductus was closed at another hospital with a single tape ligature. Within one month after the ligation, the murmur and symptoms had recurred. In August, 1950, at the age of 11, she entered Stanford Hospital for religation. The only complaint was shortness of breath on exertion. Upon physical examination, she appeared to be of normal weight and development for her age. There was moderate cardiac enlargement (Fig. 10), and a loud continuous murmur could be heard to the left of the sternum at the second interspace. The blood pressure was 112/54, the pulse rate 92. On Aug. 24, 1950, the ductus was exposed through the old incision in the third intercostal space anteriorly. There was dense scar tissue surrounding the ductus which was isolated with considerable difficulty, and a single ligature was passed around it for subsequent ligation. In attempting to free the displaced adherent vagus nerve from the scarred anterior surface to permit the application of a second ligature, the ductus was suddenly entered with profuse hemorrhage. It was evident that the nerve had formed part of the wall of the ductus. While bleeding was being controlled by finger pressure, a Potts serrated clamp was applied to the aortic arch proximal to the ductus, and the accidental rent was closed by several interrupted silk sutures. Three braided silk ligatures were then applied to tie off the ductus, division being thought inadvisable. On discharge from the hospital, August 31, a blood pressure of 130/100 and a pulse rate of 88 were recorded. Six weeks later chills and fever developed, and the murmur to the left of the sternum reappeared, at first of mild intensity but becoming increasingly loud under observation. The blood pressure was 104/60, pulse rate 120. A blood culture yielded a *staphylococcus albus,* which was resistant to penicillin, streptomycin, Aureomycin, terramycin, and to some extent Chloromycetin. The patient was treated with chloramphenicol and the cultures became sterile for a few days only, when the organisms reappeared. During this period she was febrile, obviously ill, had repeated epistaxis requiring nose packing, and looked drawn and pale. Nearly all her hair fell out. A course of bacitracin and penicillin was given without effect upon the blood stream infection.

On Jan. 11, 1951, the ductus area was again exposed through a posterolateral incision in the fourth interspace. The dissection at this time was tedious and time consuming, the entire operation lasting nine hours. After considerable difficulty, a bilobed aneurysmal swelling involving the anterior wall of the ductus was partially isolated (Fig. 11). Arterial blood could be seen coursing through it with each systole. The arch of the aorta, the left subclavian artery, and the pulmonary artery distal to the aneurysm were completely mobilized. The pericardium was opened, and a Crafoord clamp was applied to the left main pulmonary artery within the pericardium. Another clamp of the same type was applied to the distal pulmonary artery. A large side-occluding clamp of the Potts type was applied to the aorta. The ductus and the aneurysm having been completely isolated from the systemic and pulmonic circulations, the ductus was divided at the site of the aneurysmal dilatation. Two braided silk sutures were found lying within the lumen, embedded in vegetations on the ductus wall, and surrounded by granulation tissue. These foreign bodies undoubtedly accounted for the stubborn persistence of the circulating organisms. The aneurysmal wall and surrounding scar were resected; the openings into the aorta and pulmonary artery were closed with arterial silk, it being necessary to reconstruct partially the pulmonary artery. Adjacent scar tissue was used to reinforce the suture lines. Cultures taken from the vegetations grew the same organisms as had been present in the blood. All postoperative blood cultures were negative, and the patient made an uneventful recovery. A blood pressure of 112/88 was recorded on the evening of operation and has remained normal since that time. The patient has regained normal strength and health and has returned to school.

Of equal interest was the occurrence of an aneurysm of the pulmonary artery[15, 16] located exactly opposite the entrance of the ductus. This aneurysm was also the result of an endarteritis, and a stubborn blood stream infection.

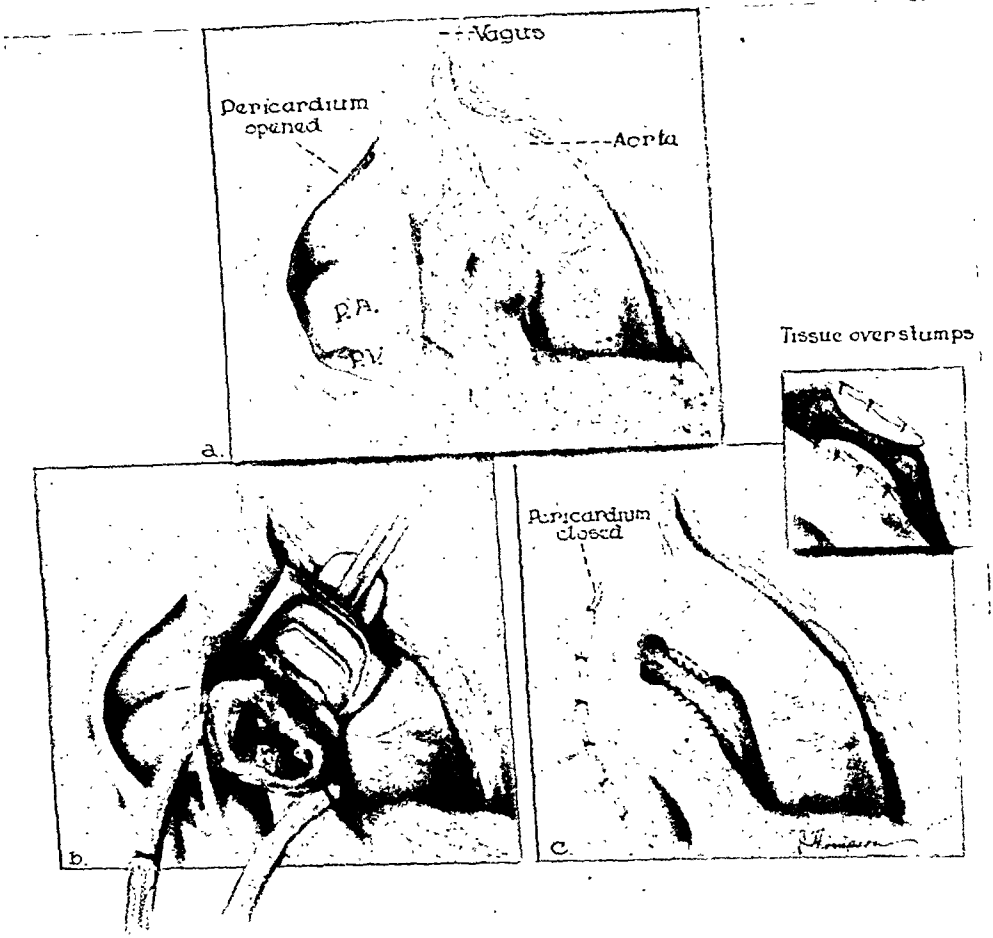


Fig. 11 (Case 4).—*a*, Biloculate aneurysm of patent ductus which followed triple ligation of ductus, and a blood stream infection with *Staphylococcus albus* which failed to yield to any antibiotics. *b*, Following complete isolation of the aneurysm the ductus was divided at the site of aneurysmal dilatation. Two silk ligatures found imbedded in vegetations were removed. *c*, Opening in the aorta was closed by a continuous suture, pulmonary artery reconstructed from ductal wall. Following operation blood stream was sterile.

CASE 5.—L. P., aged 14 years, entered the hospital with a diagnosis of congenital heart disease. A murmur had been present since birth. During her early childhood she was normal in all respects, except that she tired easily. Three years before entry she began having fainting spells when she was tired or nervous. About this time also she had what was called influenza, which was followed by a persistent fever which was found to be due to a blood stream infection. She entered a hospital where she remained for five

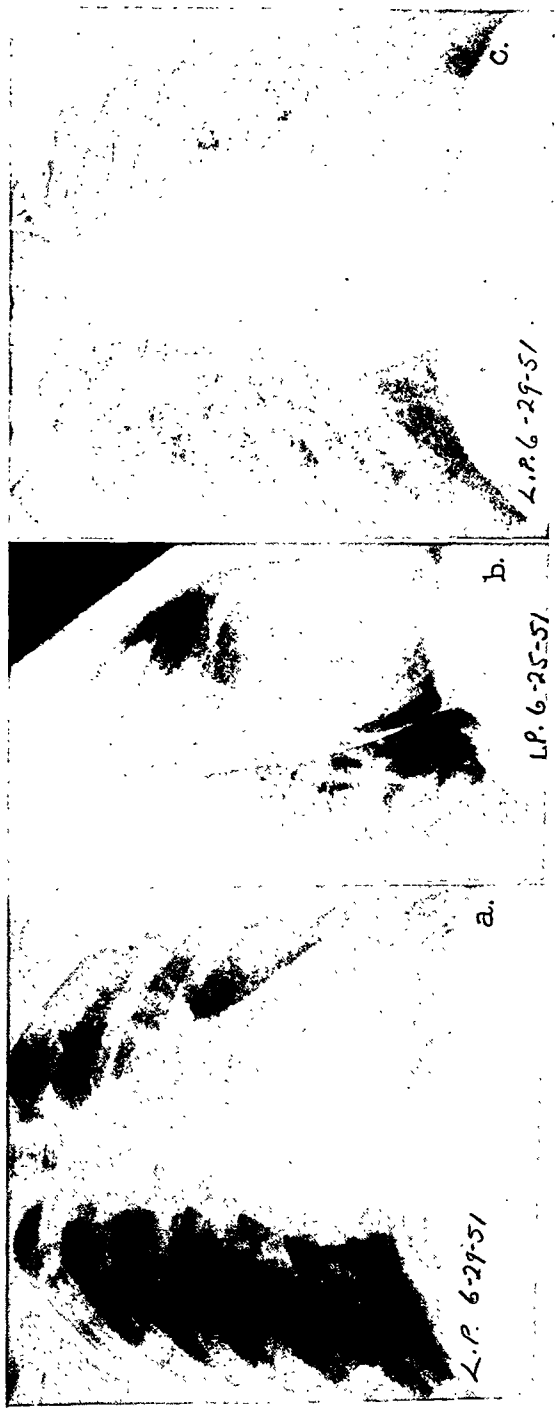


Fig. 12 (Case 5).—*a* and *b*, Roentgenograms revealed aneurysm of pulmonary artery. *c*, Angiogram revealed the aneurysm only partly opacified indicating the presence of a thrombus.

months and received 992 injections of penicillin! Following this illness she was fairly well. Upon examination she was a normally developed girl whose abnormal physical findings were limited to the heart. The blood pressure was 120/60. The heart was slightly enlarged. There was a continuous murmur to the left of the sternum in the second interspace. Roentgenograms (Fig. 12) revealed an aneurysmal swelling either of the left pulmonary artery or of the ductus. Cardiac catheterization was performed, with the findings as shown in Table I.

TABLE I

SITE OF CATHETER		OXYGEN ANALYSIS VOLUMES PER CENT
Superior vena cava		13.3
Right ventricle		13.5
Pulmonary artery		15.4
Femoral artery		17.4
PRESSURE MEASUREMENTS		
Pulmonary artery	48/18	
Right ventricle	50/6	
Femoral artery	113/65	

Angiocardiograms (Fig. 12) revealed partial opacification of the aneurysm and reopacification of the pulmonary vessels, indicating a left to right shunt. On July 7, 1951, the ductus was exposed through a posterolateral incision in the fifth interspace (Fig. 13, *a*). The ductus measured 1 by 0.7 cm. The pulmonary artery opposite the entry of the ductus was greatly dilated, forming an aneurysm which measured 4 cm. in diameter. The aneurysm contained a thrombus as was suggested by the roentgenograms. The ductus was



Fig. 13 (Case 5).—*a*, Large 4 cm. sized aneurysm of pulmonary artery opposite opening of patent ductus. *b*, Ductus closed by multiple ligations; aneurysm surrounded by irritating type of Cellophane. Patient is well.

obliterated by the ligature-transfixion method, and the aneurysm itself was wrapped with two layers of cellophane (Fig. 13, *b*), the inner layer being of the irritating type, and the outer, the nonreacting type. Her postoperative recovery was uneventful. On August 28, a blood pressure of 110/80 and a pulse rate of 78 were recorded. She now attends school

and participates in moderate athletics, but still has fainting spells occasionally. Roentgenograms reveal no change in the size of the aneurysm.

A pair of identical twins (Fig. 14) presented almost identical clinical pictures with this important difference: at operation one ductus measured one millimeter greater in diameter and this twin showed greater impairment of circulation as judged by a greater dilatation of the heart, and a lower diastolic pressure.

CASE 6.—Marjorie J., aged 3½ years, had a birth weight of 2 pounds, 13 ounces and was an incubator baby for six weeks. Her development was slow; she walked at 18 months and thereafter fatigued easily in play. A murmur was first noted at 2 weeks of age but there was no cyanosis. She was admitted for ligation of ductus on Dec. 19, 1950. On physical examination the heart was large (Fig. 15, *b*), a loud machinery-like continuous murmur was transmitted into neck and back. The pulse rate was 94, blood pressure in left arm, 102/0, right leg, 132/20; red blood cells 4,800,000, hemoglobin 14.5 Gm. per cent, white cells 9,600, 42 per cent neutrophils, 46 per cent lymphocytes, 2 per cent eosinophils, 2 per cent basophils, 8 per cent monocytes. At operation, the ductus was 18 mm. long and 10 mm. in diameter at the aortic end, 8 mm. at the pulmonic end. The blood pressure was elevated immediately following operation (Fig. 16, *A*), and the patient has remained well.

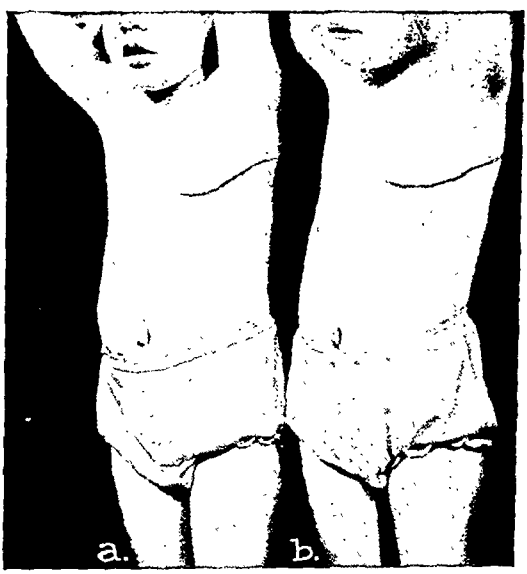


Fig. 14 (Cases 6 and 7).—Identical twins with identical patent ductus who showed almost identical clinical pictures, with identical responses following operation (see Figs. 15 and 16).

CASE 7.—Mary J., age 3½ years, had a birth weight of 2 pounds, 14 ounces and was an incubator baby for six weeks; she walked at 17 months. She was admitted for ligation of the ductus on Dec. 19, 1950. The heart was enlarged (Fig. 15, *a*), but less so than that of her twin sister. A loud machinery-like continuous murmur was heard best in the second interspace on the left but was transmitted over the whole precordium; the pulse rate was 110, blood pressure in the left arm, 90/40; in the left leg, 104/46; red blood cells numbered 5,300,000 hemoglobin 14.5 Gm. per cent, white cells 5,600, 40 per cent neutrophils, 2 per cent eosinophils, 1 per cent basophils, 50 per cent lymphocytes, 7 per cent monocytes. At operation, the ductus appeared exactly similar to that of her twin though slightly smaller; it was 18 mm. long, 9 mm. in diameter at the aortic end, and 7 mm. at the pulmonic end. The blood pressure was elevated abnormally immediately after closure of the ductus but fell to 110/80 on discharge (Fig. 16, *B*). She has remained well.

Religation of a Recurrent Ductus: Religation[11] was necessary in our very first patient operated upon in December, 1939, when the ductus was closed with a single ligature, that being the procedure then in vogue. At reoperation, the ductus was found as large as at the first operation, with only small masses of scar tissue present to disclose the site of the previous ligation. The ductus was isolated without difficulty and tied at both aortic and pulmonic ends with a 3 mm. wide tape. In view of the almost normal appearance of the ductus at this

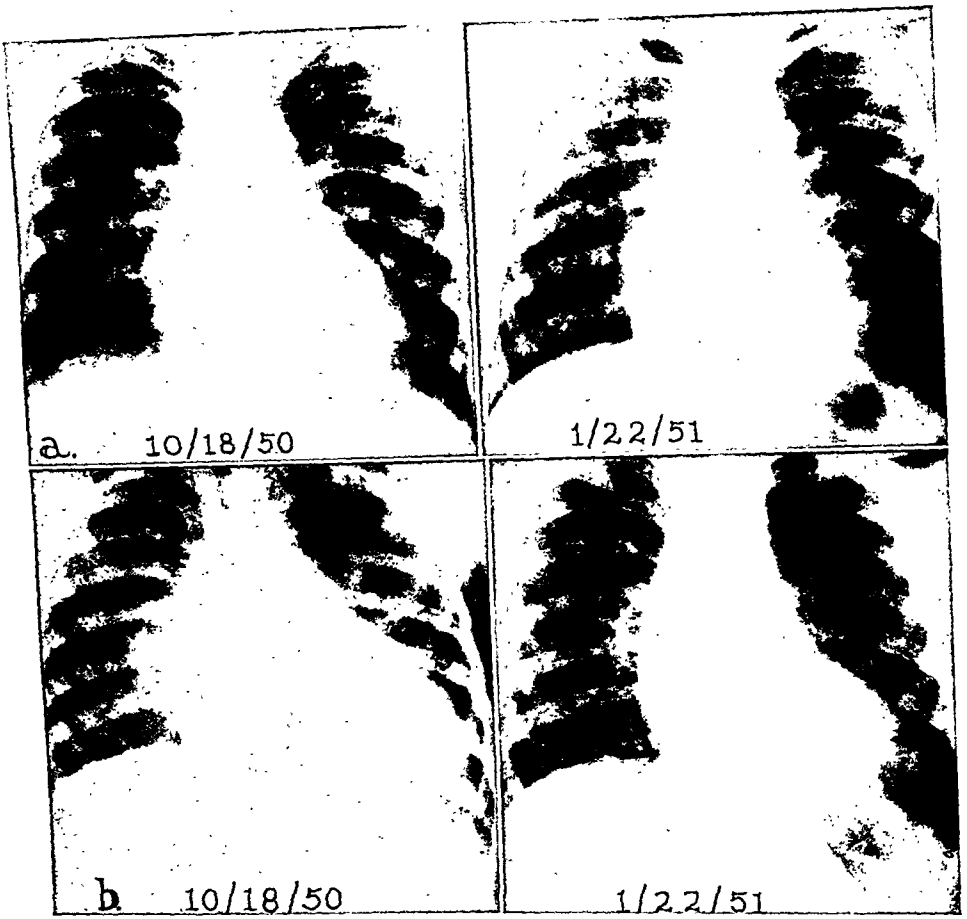


Fig. 15 (Cases 6 and 7).—Roentgenograms of chests of identical twins, *a* and *b*, before and after triple ligation of patent ductus on Dec. 19, 1950, showing decrease in size of heart mainly due to a smaller left ventricle. Note decrease also in hilar markings. In twin *b* whose heart was definitely more dilated, the ductus measured 1 mm. larger in diameter.

second operation, our medical colleagues questioned whether the ductus had actually been ligated at the first operation. Unequivocal evidence that the ductus had been properly occluded was available from the operative records. At the operating table, the blood pressure rose immediately after this first ligation from 110/70 to 130/100 and the pulse rate dropped from 100 to 70, a phenomenon directly attributable to the increased blood volume in the presence of a ductus and to the increased peripheral resistance imposed by closure of the

ductus. On December 19, ten days after ligation, the blood pressure was 120/90 as compared with a preoperative reading of 130/70. On May 20, 1940, when the murmur was again heard in full force indicating reopening of the ductus, the blood pressure was 124/60. At the second operation on April 5, 1941, closure of the ductus was followed immediately by a rise in pressure from

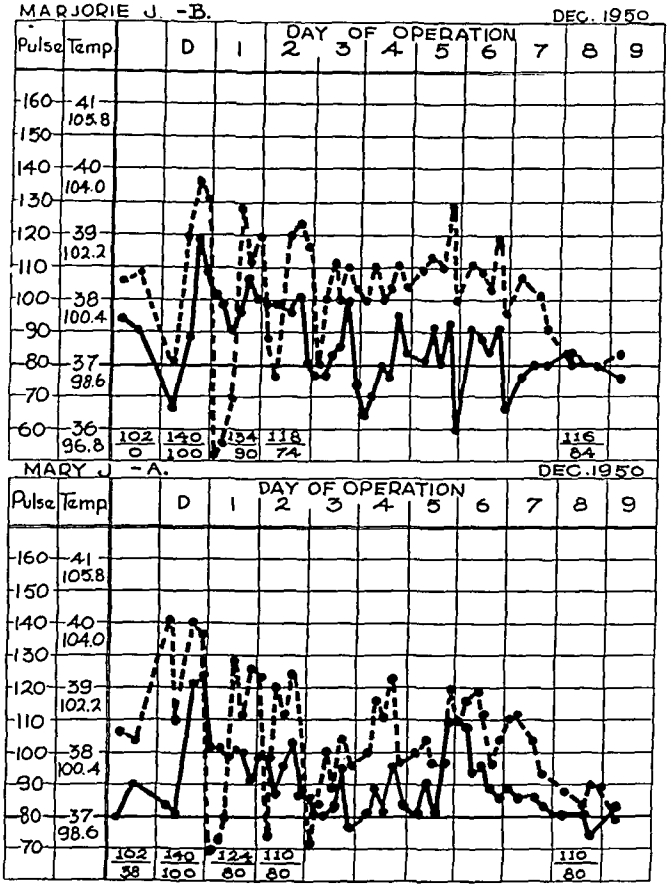


Fig. 16 (Cases 6 and 7).—Temperature and pulse chart of twins, *a* and *b*, showing almost identical response to closure of ductus. Note increase in blood pressure from 102/0 to 140/100 on day of operation in twin *b*, and from 102/38 to 140/100 in twin *a*.

128/50 to 148/106 and by a decrease in pulse rate from 116 to 100. On discharge from the hospital, the pressure was 112/84, and this normal pressure has been maintained. The patient has married and has had several children.

At the operating table, this evidence of an increase in blood pressure on trial closure of the ductus by digital pressure is reassuring evidence, first, that the proper structure has been isolated and, second, that its permanent closure can be tolerated with impunity without depriving the patient of a shunt necessary for proper aeration of the blood as might occur in connection with a patent ductus accompanying a mild, unrecognized tetralogy of Fallot.

The fortuitous passing of a catheter from pulmonary artery through the ductus into the aorta is worthy of mention to illustrate the effect of a ductus upon oxygen values at various sites.

CASE S.—L. P., was 6 years old. The mother had had rubella at one month of gestation. Murmur was first noted in the infant at 10 weeks of age. At 2½ years, a thoracic deformity (scoliosis) was first noted. The patient was admitted to the hospital in September, 1949, for cardiac catheterization studies, there being a question of an interauricular



Fig. 17 (Case S).—Roentgenogram revealed catheter looped through patent ductus into the aorta.

septal defect. A cardiophonogram recorded a prominent continuous murmur in the pulmonic area, and a systolic and diastolic murmur at the apex, which was thought to be the same murmur present in the pulmonic area. The roentgenogram of the chest was interpreted as showing both right and left ventricular enlargement and marked enlargement of the pulmonary vascular bed.

Cardiac catheterization resulted in threading the catheter from the pulmonary artery into the aorta (Fig. 17).

TABLE II

SITE OF CATHETER	OXYGEN VOLUMES PER CENT	PRESSURE MEASUREMENTS
Superior vena cava	10.9	
Right auricle	10.8	
Right ventricle	10.5	5.5 mm. Hg
Pulmonary artery	13.4	61/11
Aorta	13.4	60/37
Oxygen capacity	15.0	84/45
Oxygen saturation	89%	

On Nov. 9, 1949, the patent ductus was closed by multiple ligations. The ductus measured 1.4 cm. in diameter. Before ligation of the ductus, the pulmonary conus had a diameter of 2.3 cm., after ligation, 1.5 cm. The blood pressure rose to 120/80 immediately after ligation and the following day was 130/110, but subsequently dropped again to 104/80.

Pregnancy as a complication of a patent ductus is illustrated by the following patient:

CASE 9.—Mrs. O. M. N., 26 years of age, was known to have had a heart murmur since the age of 8 years. Although she had been followed by a competent pediatrician before that time, no murmur had been noted, and after its discovery it apparently was rather evanescent. She played basketball in high school without discomfort. Since an attack of measles one year before admission, there had been some shortness of breath, which had become very distressing since becoming pregnant about a month before admission. Formerly a choir singer, she could no longer sing due to dyspnea. The least effort at work tired and distressed her. Her heart "pounded" on excitement. There had been no cyanosis, no fever, and no swelling of the ankles.

Physical examination disclosed a machinery-like continuous murmur over the second interspace to the left of the sternum, the systolic phase audible over the whole precordium; blood pressure 140/70, pulse rate 120, after exercising, 148. The electrocardiogram was normal; red blood cells numbered 3,500,000, hemoglobin 80 or 13 Gm. per 100 c.c., white cells 5,100. On March 18, 1948, the ductus was divided and the two cut ends were closed with continuous sutures. On closing the ductus, the pressure rose from 110/70 to 110/90. Recovery was uneventful and she left the hospital on March 28, only to lose the 1-month-old fetus three days after returning home. Blood pressure one month later was 136/84.

The case is cited to illustrate the baneful effect of pregnancy on a heart already burdened by a patent ductus.[17] The increased total blood volume[18] and increased cardiac output which occur in pregnancy together with the increased output and increased blood volume due to the ductus both combined to increase the cardiac burden and the dyspnea that developed during the pregnancy. The diminished cardiac reserve imposed by a ductus in adult years as observed in this pregnant woman provides additional justification for advising closure of the ductus in childhood.

Five patients were explored by thoracotomy for symptoms and signs indistinguishable from those of an open ductus, but no ductus was found.

CASE 10.—V. M., 8 years of age, had a cardiac murmur discovered at the age of 26 months. She had been on restricted activity, but had few, if any, cardiac symptoms. Upon physical examination, there was a harsh systolic murmur over the precordium, loudest over the second and third interspace to the left of the sternum. A thrill was present in this region. Posteriorly, the murmur could be heard but it was less loud. The blood pressure was 112/72. An electrocardiogram showed right axis deviation. Roentgenograms revealed a greatly enlarged pulmonary conus. This was confirmed by angiocardiograms which were interpreted as showing reopacification of the pulmonary artery from the aorta, suggesting a patent ductus. On April 19, 1941, the left thorax was explored through an anterior incision. A greatly enlarged pulmonary artery was found, and at its origin there was a marked thrill and a loud bruit heard through a sterile stethoscope. Pressure could not completely obliterate it, although it could be reduced in intensity. No ductus was found. A diagnosis of aortopulmonary artery septal defect was made, closure of which was not considered feasible because of its location. She was last seen in 1945 when she was having growing pains suggesting the possibility of rheumatic fever.

CASE 11.—M. S., a 19-year-old Japanese girl, was known to have had heart trouble for five years. There had been no symptoms until one year before entry, when palpitation and exertional dyspnea developed. Upon examination, there was a very loud, continuous murmur and thrill in the third left interspace, heard loudest approximately 6 cm. from the midline. The murmur could also be heard over the whole left thorax, and was quite loud in the back. Roentgenograms showed a large heart suggestive of patent ductus. The blood pressure was 108/40. At operation, on Jan. 15, 1949, an obliterated ligamentum arteriosum was found. A marked thrill was present at the base of the pulmonary artery, just at its point of origin. Pressure in this region obliterated the thrill. It was not considered technically feasible to close the defect due to its close proximity to the heart. There has been no progression of symptoms since the operation.

CASE 12.—J. A. W., aged 6 years, was first suspected of having cardiac trouble at the age of 6 because of a persistent tachycardia and systolic murmur. Frequent colds, easy fatigability, and a bluish pallor have been noted frequently since.

Physical examination disclosed an enlarged heart, a prominent pulmonary artery, a harsh systolic murmur along the left sternal margin, and a soft diastolic murmur in the midsternal area. A blood pressure of 100/82, a pulse rate of 90, and left axis deviation were recorded.

Catheterization studies suggested an atypical interventricular septal defect but a patent ductus could not be ruled out (Table III).

TABLE III

SITE OF CATHETER	OXYGEN CONTENT VOLUMES PER CENT	PRESSURE MEASUREMENTS
Inferior vena cava	11.0	
Superior vena cava	13	
Right auricle	13.6	
Right ventricle	16.2	84/7 mm. Hg
Pulmonary artery	15.9	84/44 mm. Hg
Femoral artery	16.9	86/54 mm. Hg
Femoral artery	92% saturated	
Hemoglobin	12.8 grams	

At operation on June 22, 1950, there was no ductus present, the heart was large; the left pulmonary artery was greatly dilated with a diameter of 2.5 cm., whereas the arch of the aorta seemed hypoplastic with a diameter of only 1.5 cm. Only a mild systolic thrill was present over the pulmonary artery. A high interventricular septal defect was thought to be most probable.

CASE 13.—M. S., 25 years of age, had always been short of breath, easily fatigued, and pale since birth. Married seven years previously, there had been three pregnancies with two living children. Six months before admission to hospital, while five months pregnant, she had become more dyspneic, unable to do her work, and fainted on several occasions. In the two months before admission there had been fever, with chills, severe sweating, severe headaches, and a blood culture had yielded an organism identified later as an anaerobic, gram-positive member of the Actinomycetales group for which 200,000 units of penicillin had been administered every three hours for a week.

Physical examination showed marked pallor, dyspnea while lying in bed, visible thrust of the precordium, palpable thrill over the whole precordium, and a loud machinery-like murmur, most intense over the left third interspace, but heard over the whole precordium, in the left axilla and back and in the neck. The temperature was 38° C., the pulse rate 110, respiration 20, blood pressure 142/58; red blood cells 4.9 million, hemoglobin 75 per cent, venous pressure 10 cm. water; circulation time, arm to tongue, 34 seconds.

The clinical diagnosis by all who saw her was patent ductus with bacterial endarteritis. Under observation and with penicillin therapy, the temperature improved although it remained somewhat elevated, and the blood pressure became 160/0, suggesting an increasing shunt; accordingly early operation was advised.

At operation on Oct. 1, 1947, no ductus was found. The thrill which was greatest immediately over the origin of the pulmonary artery could be obliterated only by almost complete closure of the pulmonary artery by pressure. A high fever and rapid pulse followed the operation; the patient lost ground rapidly and died in cardiac failure six days after operation.

At necropsy, the foramen ovale measured 1.5 by 0.5 cm. with vegetations on its margin. There was an interventricular septal defect (Fig. 18) measuring 1.7 by 1.2 cm., and an aneurysm of the sinus of Valsalva providing a communication into the right ventricle, measuring 0.2 by 0.3 cm. The pulmonary artery was greatly dilated, measuring 9.8 cm. in circumference, the aorta, 6.2 cm. The tricuspid valve measured 14.7 cm. in circumference, the mitral valve, 11.7 cm. Endocarditis involved the aneurysm, the septal defects, and the pulmonary artery.

CASE 14.—A. A., 33 years of age, entered the hospital with the complaint of increasing dyspnea. There had been a heart murmur and attacks of cyanosis in infancy, but no

serious symptoms until ten years previously when an abrupt attack of palpitation occurred lasting about four hours. Examination then showed a dextrocardia, and a systolic murmur over the right precordium. During the two years before entry, he had noticed a gradually increasing fatigability and dyspnea. One year before entry he had hemoptysis and an attack of vertigo. Upon examination, the blood pressure was 120/70. The neck veins pulsated. The heart was on the right. The murmur was now clearly continuous in the right second interspace, with a rough, loud systolic component at the third and fourth right interspace. Roentgenograms showed an enlarged left ventricle and congested pulmonary vessels which pulsated actively. Electrocardiograms showed abnormal T waves and abnormal precordial leads. Laboratory and blood studies were normal. Cardiac catheterization* revealed an oxygen content of 15.7 volumes per cent in the right ventricle and 20.8 in the pulmonary artery. The femoral artery blood was 96 per cent saturated. The right ventricular pressure was 77/0, and the pulmonary artery 20/-2. These findings were considered as being compatible with patent ductus arteriosus and pulmonic stenosis. Operation was performed on April 1, 1952, through a right posterolateral incision. The

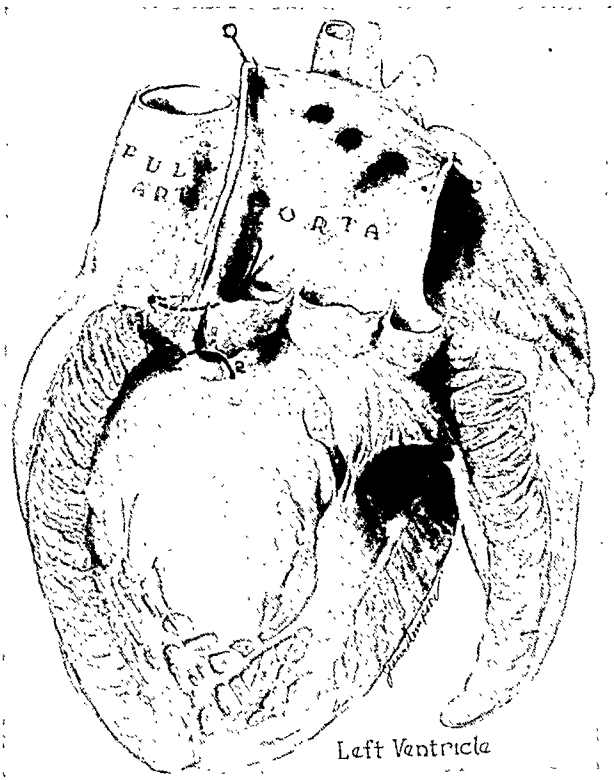


Fig. 18 (Case 13).—Following thoracotomy for presumed ductus which was not found, the patient died of bacterial endocarditis. Two lesions accounted for signs and symptoms indistinguishable from those of ductus, namely, (1) an aneurysm of the sinus of Valsalva transmitting blood from aorta into pulmonary artery, and (2) a high interventricular sepal defect.

ductus was not patent. After opening the pericardium, it was found that the maximal thrill was in the myocardium at the beginning of the aorta and pulmonary artery, giving the impression of a defect near or perhaps just distal to the valves. It was impossible to stop the thrill by pressure. Due to the proximity of the coronary vessels, it was felt inadvisable to attempt closure of the defect. The postoperative recovery was uneventful.

*Performed by Dr. Hultgren.

Operative Technique.—The following important details characterize the operation as performed at the present time.

1. Operative approach either through an anterior incision in the third interspace with division of the third and second costal cartilages, or through a posterolateral incision in the fourth interspace with separation of the ribs but without either division or excision of a rib. The former incision is preferred in boys, and the latter in girls or in young women sensitive to the appearance of a scar.

2. Identification of the site of the ductus by palpating for the area of greatest thrill.

3. Identification of the vagus nerve and of the recurrent laryngeal nerve as it runs underneath the ductus.

4. Isolation of the transverse arch proximal to the left subclavian artery for the subsequent application of a Potts serrated clamp or an occluding clamp such as the cloth-shod Crafoord clamp just prior to ligation of the ductus.

5. Isolation of the anterior, lateral, and medial walls of the ductus close to the thicker walled aortic end rather than near the pulmonic end. The transverse diameter of the ductus is measured, and this measurement is used in locating accurately the site of the posterior wall of the ductus so as to stay well away from it and thus avoid injuring it during dissection of the posterior periductal tissues. Once the tip of the right-angled clamp has penetrated the periductal tissues to reappear on the side opposite the operator, the complete isolation of the ductus is a matter of careful division and separation of tissues now easily observed and identified. The full length of the ductus having been properly developed, the effect of its closure on blood pressure and pulse is determined and measurements are taken. The decision is then made whether to apply multiple ligatures in continuity[19] or to apply four clamps, divide between the central two clamps, remove these two clamps, and suture the open cuff projecting beyond the remaining two clamps.[20] Recently, a decision to divide and suture the ductus in a 4-year-old child was made because of its large size, 1.5 cm. in diameter. To apply a ligature to such a large ductus invites the real danger of fracturing the wall of the ductus while attempting to drive home the knot. Division and suture were easily performed.

A word of caution when applying multiple ligatures is in order. In applying the first ligature to the aortic end of the ductus, the ligature is anchored in place by taking a few bites of the periductal and periaortic tissues so that in tying, the ligature will not slip from the end to the center of the ductus. Also before tying this ligature, a serrated Potts clamp[21] or a Crafoord clamp is applied to the transverse arch in the area previously mobilized to be released again as soon as the ligature has been tied. This abolishes pulsation in the ductus during the act of ligation, thus reducing the hazard of fracturing the wall of the ductus. The re-establishment of an open ductus following ligation may well have its origin in injury to its wall at the moment of tying a knot against the resistance of a vigorously pulsating blood stream. A ligature is next applied to the pulmonic end of the ductus, it also being anchored in place by taking a few bites of the periductal tissues just where it joins the pulmonary artery.

TABLE IV. AGE AT OPERATION

AGE (YR.)	NO.	AGE (YR.)	NO.
0 to 2	5	16 to 20	6
3 to 5	25	21 to 25	3
6 to 10	23	26 to 30	2
11 to 15	10	31 to 35	1

Two transfixion ligatures are then applied to the ductus in the segment between the two ligatures previously placed. The site of ligation is bathed in penicillin solution, the pleura overlying the ductus is closed, and the chest wound resutured. Intercostal drainage is rarely used. Very little blood is administered by transfusion unless real blood loss has occurred since closure of the ductus invariably causes a temporary rise in blood pressure due to the autotransfusion that occurs at the moment of ligation, attributable to the increased blood volume that has occurred in the presence of the ductus.

TABLE V. REASONS FOR OPERATING

Clinical diagnosis with few or no symptoms	30
Clinical diagnosis with enlarged or failing heart	5
Clinical diagnosis with moderate to severe dyspnea and fatigability, poor growth, or endocarditis	40

Another word of caution is in order. If, at any time, bleeding should occur during isolation of the posterior wall of the ductus and this bleeding is easily controlled by mild digital pressure, further dissection is immediately discontinued. Two courses of action are then available. If no further bleeding occurs upon release of digital pressure, several minutes later, the operation may be terminated and the wound closed. After the elapse of six months to one year, reoperation may again be considered and performed without difficulty. This procedure was employed in one case in which this mild bleeding occurred during isolation of the posterior wall of the ductus. Disaster might have followed if the operation had been pursued further. Six months later complete success marked the second operation and the child is now well. The only death occurred in a young 7-year-old patient in whom this precaution was not observed when slight, easily controlled bleeding occurred while isolating the posterior wall of the ductus. In pursuing the dissection further, excessive bleeding was encountered, requiring transfusion of 2 liters of alien blood (presumably properly matched) and 2 liters of her own blood recovered at the operating table. De-

TABLE VI. ASSOCIATED NONCARDIAC ANOMALIES

Cataract and strabismus*	1
Cataract*	1
Cataract and clubfoot	1
Cataracts, bilateral, internal strabismus, thoracic deformity, displaced lenses*	1
Cataracts, nystagmus†	1
Strabismus	1
Undescended testes	1
Hypogonadism	1
Megacolon	1
Thoracic deformity*	1
Harelip, cleft palate, syndactylism	1

*Mother had rubella at about one month of gestation.
†Mother had rubella at two to three months of gestation.

spite the successful division of the ductus, complete anuria followed and she died in convulsions seventy-two hours after operation.

A second course of action would be to continue the operation by isolating all structures proximal and distal to the ductus, as described in Case 4, and to proceed with division of the ductus. This decision may, in some measure, depend upon the experience of the operator.

Tables IV to VI provide some pertinent details.

SUMMARY

The ductus arteriosus with or without symptoms should be closed by operation if it remains patent beyond the age of 5, in order to avoid the hazards of retarded physical development, cardiac decompensation, or bacterial endocarditis.

A patent ductus is in effect an arteriovenous fistula within the heart and has the same effects upon the circulation as a peripheral arteriovenous fistula including increased cardiac output, limited in the case of a ductus to one-half of the heart, increased blood volume, reduced blood pressure, particularly diastolic pressure, cardiac dilatation, and eventually decompensation.

Cardiac failure may occur at an early age if the ductus is large and easily distensible. As the child with a patent ductus grows to adult stature the lower resistance imposed by the pulmonary circulation as compared with that of the systemic circulation will attract a progressively greater volume of blood into the shorter circuit, resulting in progressive dilatation of this shorter circuit including the ductus and the left heart. This progressively increasing blood flow through the shorter circuit is limited by two factors, the innate rigidity of the ductal wall itself and the increasing fibrosis of the pulmonary capillaries incident to their connection with the high pressure of the systemic circulation. These two factors diminish the difference in the resistances of the two systems and reduce, and may even abolish, the tendency for an increasing volume of blood to flow through the ductus, the effects of which then remain stationary. Both factors are operative in delaying the appearance of cardiac symptoms to adulthood, but the appearance of cardiac symptoms is inevitable if a difference in the resistances of the two systems persists, if the ductus remains open, and if its walls remain distensible. Just as a dilating peripheral fistula eventually results in cardiac decompensation, so will a dilating ductus increase the flow through it and produce left heart failure.

Bacterial endocarditis and endarteritis occurred in one patient at the early age of 7, cured by ligation of the ductus. An aneurysm of the ductus occurred in another patient of 11 years whose ductus had been ligated twice previously and was accompanied by a bacteremia completely resistant to all antibiotics. Division of the ductus at the site of the aneurysm resulted in immediate disappearance of the bacteremia and complete restoration to health.

In another patient of 14 years of age, an aneurysm of the pulmonary artery opposite the entrance of the ductus followed a bacterial endarteritis for which 992 injections of penicillin had been given with control of the bacteremia. Ligation of the ductus and wrapping of the aneurysm with Cellophane has stayed the aneurysmal process.

A pair of twins, 3½ years old, showed about the same clinical response to patent ductus both before and after an operation in the course of which remarkably similar ductus in shape and size were found. The circulatory effects of one ductus were slightly more pronounced as shown by a larger heart and a lower diastolic pressure. At operation this ductus measured one millimeter larger in diameter at both the aortic and pulmonic ends.

One patient, cyanotic in life, was not operated upon due to a mistaken diagnosis, and died of cardiac failure at the age of 16 years. Necropsy revealed evidence that the flow of blood through the ductus throughout life had been from pulmonary artery to aorta with an excessive development of the right heart as compared with the left. It is questionable if the patient could have survived ligation of the ductus at this age because of the preponderant hypertrophy of the right heart. Recognition of this condition earlier in infancy would no doubt have permitted permanent correction by operation.

A woman 26 years of age with a known patent ductus but without serious symptoms for many years became pregnant. At the end of about a month, increasing dyspnea and easy fatigability led to division of the ductus, which eliminated all cardiac symptoms. This experience provides added support for early operative closure of a ductus no matter how symptomless it may be in childhood.

Five patients with all the signs and symptoms of patent ductus were operated upon by thoracotomy but no ductus was found. One patient, a married woman, 25 years of age, died following thoracotomy with advanced signs of bacterial endocarditis. At necropsy, a high interventricular septal defect with an aneurysm of the sinus of Valsalva communicating with the pulmonary artery, complicated by the extensive vegetations of a bacterial endocarditis, was found. The other four patients recovered without accentuation of symptoms and were presumed to have aortic-pulmonic septal defects or high interventricular septal defects involving both aorta and pulmonary artery.

Eight patients with patent ductus were treated by division and sixty-seven by multiple ligation in continuity. The first ductus operated upon recurred following ligation with one ligature, and one ductus recurred following triple ligation with the formation of an aneurysm. Both patients were cured, the first by religation, the second by division of the ductus at the site of the aneurysm. One death, in seventy-five cases, occurred on the third day following operation for division of the ductus due to a complete anuria incident to blood transfusion.

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DISCUSSION

DR. RICHARD H. MEADE, Grand Rapids.—I have no intention of discussing this excellent paper as I am not qualified to do so, but I do wish to report an interesting experience that my associate, Dr. Rasmussen, had two years ago, as it may be of value to some of you.

The patient was a woman in her 20's who had an obvious patent ductus arteriosus. At operation, during the dissection about the duct, serious bleeding was encountered which necessitated ligation of the pulmonary artery in order to control it. The ligation was done without fear because we knew that it had been demonstrated that it produced no deleterious effects. A few days later we left for the Denver meeting of this Association with no worries about the patient. Upon our return, however, it was evident that she had gangrene of the lung and a pneumonectomy was done. She died at the conclusion of the operation. Autopsy failed to demonstrate any abnormalities that could have accounted for the unusual course of events.

It is our belief that the gangrene developed because this patient's lung had been supplied for some twenty years with arterial blood through the ductus, as well as through the bronchial arteries. The bronchial arteries had, therefore, not developed normally and when the entire load of the circulation had been thrown on them by ligation of the pulmonary artery they had been unable to meet the demand. It is likely that an infant could tolerate ligation of the pulmonary artery, but we believe that should it ever become necessary to ligate the pulmonary artery in an adult with a patent ductus arteriosus, then the lung also should be removed.

DR. EDGAR W. DAVIS, Washington, D. C.—I should like to make a few comments on the presentation of Drs. Holman and Gerbode, which I have enjoyed immensely. Upon many occasions, at the meetings of this Association, when discussing the technical procedure of double ligation versus division and suture of patent ductus arteriosus, I have defended the former method. Several years ago, when I discussed papers by Drs. John Jones, Robert Gross, and Clarence Crafoord, I reported a series of twenty-five or more patients upon whom I had operated, using the multiple ligature method, and I stated at that time that we had had no recurrences and no serious complications. On that basis, I saw no reason to change to the technique of division and suture.

Now, after a period of additional years, with accumulated experience involving more than a hundred cases, I find that we have two definite cases of recurrence or recannulization, and possibly a third case which at this time has not been proved. The patent ductus clamp designed by Dr. Willis Potts has made the operation of division and suture one of complete safety. As a matter of fact, out of our first sixty-five patients operated upon, we did double ligations in sixty-three and divided and sutured in only two. Because of the fact that the operation can now be performed with safety with the use of the Potts clamp, thereby minimizing the percentage of recurrence, I wish to take this opportunity to retract my previous statements in which I advocated double ligation instead of division and suture. So, in the words of the famous poet, Edgar Allen Poe—

"Be not the first by whom the new is tried,
Nor the last to lay the old aside."

DR. HARRIS B. SHUMACKER, JR., Indianapolis.—The fascinating paper of Drs. Holman and Gerbode has raised a great many interesting problems. I propose to discuss only one of them, that of right ventricular hypertension in cases of patent ductus arteriosus. We have had three patients whose disorder fell into this category. In one child the right ventricular pressure was approximately the same as the systemic arterial pressure. Unfortunately, in this case studies were not complete enough to permit us to say whether there was reversal of flow through the ductus during any phase of the cardiac cycle. At the time of operation the ductus was dissected free and clamped cautiously. Marked tachycardia developed and the clamp was released. On several successive occasions clamping the ductus brought about the same alarming increase in cardiac rate. Finally, however,

the ductus could be clamped without change in rate or rhythm of the heart. The ductus was consequently divided. During the process of suturing the divided ductus the patient developed auricular fibrillation. Fortunately she withstood the operation without difficulty. Shortly afterward conversion to a normal rhythm was achieved, and she has remained quite well.

In one young woman in her late 20's there was good evidence of reversal of flow through the ductus from cardiac catheterization studies and from study of arterial oxygen saturation in upper and lower extremities. Because we were informed of several disastrous attempts of others to treat cases of this sort surgically, we elected not to operate upon this patient.

From cardiac catheterization, a third patient, also in her late 20's, was known to have a large atrial septal defect in addition to a patent ductus. At times the right ventricular and pulmonary artery pressures were higher than that of the aorta and there was good evidence of reversal of flow through the ductus during some phase of the cardiac cycle. There was additional evidence of some unexplained vascular shunt in the lungs. Because of this fact and the presence of an atrial septal defect, it was thought that she might withstand division of the ductus. The hazards of operation were explained to the patient and she desired to have us proceed with it. When the ductus was dissected free it was clamped with caution. There was no change noted in cardiac rate or rhythm, arterial or venous pressure, and there was no evidence of pulmonary edema. Accordingly, the ductus was divided. Her convalescence was uneventful. Postoperative catheterization studies reveal the right ventricular and pulmonary artery pressures still to be very high. In contrast to the situation before operation there is now evidence of a left to right ventricular shunt. It thus appears that this patient had a ventricular septal defect in addition to an atrial septal defect and a patent ductus arteriosus. She apparently has a complicated and belatedly recognized Eisenmenger complex. If there has been any change since operation it has been one of mild improvement.

DR. PAUL T. DE CAMP, New Orleans.—I should like to refer to two patients with persistent patent ducti, pulmonary hypertension, and a partial reversal of blood flow through the ducti. To date, there is no adequate explanation for the increased intrapulmonary resistance and the pulmonary arterial hypertension which develops in some patients with patent ducti. There are a few reported cases, similar to the one reported by Dr. Holman, in which there finally develops a complete reversal of flow with generalized cyanosis. In the presence of significant pulmonary hypertension the clinical diagnosis of patent ductus arteriosus is difficult, because the diastolic phase of the typical continuous murmur tends to disappear. The diagnosis may be clarified by retrograde aortography and cardiac catheterization. In our opinion, as long as the degree of shunt from the aorta to the pulmonary artery exceeds the shunt from the pulmonary artery to the aorta, the patient may be considered as a candidate for ductal obliteration. The necessary information may be computed from the determination of the cardiac output and the oxygen content of simultaneous blood samples obtained from the right brachial, a femoral, and the pulmonary artery.

One patient, an 11-year-old girl, had had serious cardiac symptoms for several years. Congestive failure was controlled with difficulty. The clinical diagnosis was difficult, and the diagnosis was established only by retrograde aortography and cardiac catheterization. The pulmonary arterial pressure equalled the aortic pressure. The left to right shunt slightly exceeded that from right to left. At operation, fifteen minutes after the application of clamps to the ductus, a peculiar type of cardiac arrest occurred. Regular auricular and ventricular contractile movements continued but no blood was expelled from the heart because diastolic relaxation did not occur. Cardiac massage was ineffective. Normal cardiac activity resumed after six minutes and the ductus was quickly ligated, but the child died six hours later from the results of cerebral damage.

A 32-year-old man has had known heart disease all his life with reduced activity and retarded physical development. Hemoptysis was present for ten years and culminated after

hospital admission in massive hemoptysis of 1800 c.c. of blood in a twenty-minute period. Pulmonary hypertension, a patent ductus, and pulmonary arterial sclerosis were diagnosed on clinical, electrocardiographic, and roentgenologic examinations. Following transfusion therapy and the demonstration, on bronchoscopy, of a blood clot protruding from the right middle lobe orifice, an emergency right middle lobectomy was performed. A pulmonary arterial aneurysm 1.5 cm. in size was found, which had ruptured and filled the entire lobe with blood. The patient has recovered from the lobectomy. Studies of the degree of the shunt through the ductus have not been completed, but we have evidence of a partial reversal of blood flow.

DR. HOLMAN (Closing).—I am very grateful for these very fascinating additional aspects of this problem, particularly Dr. Shumacker's remarks. I would like to say that when we ligate the ductus we do not rely on two ligatures. We try to emulate Dr. Blalock by applying two ligatures at either end of the ductus and two transfixing ligatures between them, with frequently a fifth at the center of the ductus. I am sure that two ligatures will not suffice. We have divided the ductus in eight cases. We recently divided the ductus in a child aged 6, who had a ductus 1.5 cm. in diameter. Ligatures around such a large ductus can easily cause trouble with fracture of its walls. But we are content in most instances to do the quadruple or quintuple ligation.

The problem that Dr. Shumacker raises is, of course, an interesting one, and it shows the value of first attempting to find out by digital pressure on the ductus, whether the patient will tolerate ligation of the ductus, or not. Occasionally, a ductus may be encountered which provides flow into the lung in a mild case of tetralogy of Fallot, and under these circumstances one would not want to ligate it. This question of tachycardia that yielded eventually to closure of the ductus, probably has to do with reversal of flow through the ductus. Of course when we have increased pressure in the pulmonary system incident to a direct connection with the aorta, pulmonary fibrosis and pulmonary hypertension is the inevitable result, and it may be that peripheral resistance in the two systems will eventually equal each other, with the result that flow through the ductus will alternate, first in one direction and then in the other, depending upon such other factors as complete rest or violent exertion.

THE ANATOMIC (HISTOLOGIC) BASIS AND EFFICIENT CLINICAL SURGICAL TECHNIQUE FOR THE RESTORATION OF THE CORONARY CIRCULATION

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PHILADELPHIA, PA.

THE principle of arterialization and of the reversal of the flow of blood through the coronary sinus and its ramifications was first suggested by Roberts[1] and was first successfully carried out in both the experimental laboratory and in human clinical cases by Beck and his co-workers.[2-5] It is the purpose of this article to confirm the beliefs of Beck and associates and to establish the anatomic basis for the clinical benefits observed. A relatively minor technical modification of the original Beck operation is offered which is believed to lessen the likelihood of thrombosis and obliteration of the arteriovenous anastomosis.

The chief field of applicability of this procedure is found in the treatment of occlusive disease of the coronary arterial system (atherosclerosis). However, arterialization of the coronary sinus might well prove useful in individuals presenting the anomaly of one or both coronary arteries (especially the left) arising from the pulmonary artery.

Coronary occlusion, myocardial infarction, angina pectoris, and acute coronary insufficiency are all essentially manifestations of the same underlying process—arteriosclerosis affecting the coronary arterial system. Like arteriosclerosis elsewhere, it may be part of a generalized process involving all of the arteries of the body in approximately equal degree, or it may be so much more advanced and progressive in the coronary arteries than elsewhere as to seem to be an isolated process. It is essentially these latter cases which offer the best indications for surgery and the best clinical results.

From the clinical standpoint, one may consider coronary arteriosclerosis to be essentially an occlusive process in which the lumina of the large and of the medium-sized coronary arteries are progressively diminished by subintimal thickening of the vessel walls. Plaques of subintimal cholesterol deposition or of actual ulceration may be seen predisposing to the development of thrombosis with complete obliteration of the lumen (Fig. 1). Since the muscle-bearing moderate-sized branches of the coronary arteries are functionally similar to end arteries for a given area of the heart, the segments of myocardium normally supplied by each of these vessels may become infarcted unless sufficient collateral channels have become established or have opened up to maintain nourishment of the myocardium. Schlesinger and Zoll[6] have

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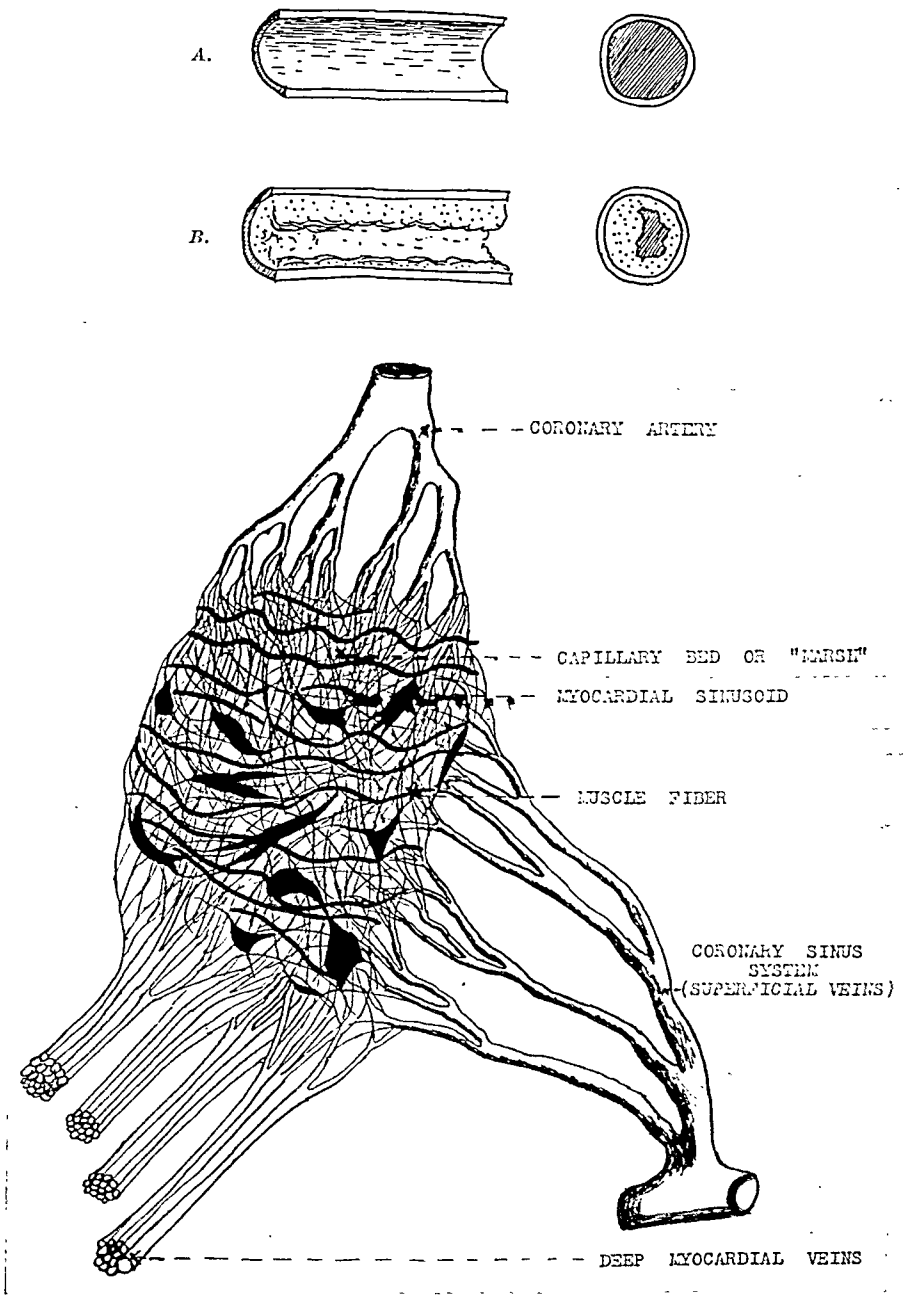


Fig. 1.—*A*, Diagrammatic drawing of a normal coronary artery. *B*, Diagrammatic representation of the diminished lumen in a coronary artery produced by subintimal thickening of the vessel walls. *C*, Concept of left ventricular myocardium as a "marsh" fed by branches of the coronary arteries. After depletion of its oxygen, 40 per cent of the blood is drained off by the deep myocardial veins and 60 per cent is drained by the "expendable" coronary sinus system. The latter therefore offers the surgeon an available, even more extensive system of blood vessels capable of transporting arterial blood to the capillary bed in cases with diminution in coronary arterial flow.

shown that only the larger and medium-sized branches of the coronary arteries are affected by this process, the smaller branches without muscular coats being spared. Hence there are some possibilities for the development of an adequate collateral blood supply through these smaller branches. However, the development of such collateral circulation (intercoronary communications) is often a slow process and is inadequate to protect against the effects of a sudden complete occlusion of one of the coronary arteries.

At best, the development of such collateral circulation between the various segments of the coronary arterial system is unpredictable, due to the marked variation in the vascular pattern in different individuals. It can improve the distribution of the blood provided by the major coronary arteries, but it cannot significantly increase the total amount of blood available for myocardial metabolism and nutrition if there is appreciable diminution in this central coronary supply. Hence all operative and medical mechanisms which rely for their effects upon the development or enhancement of normally minimal intercoronary blood flow are limited in their ultimate clinical potentialities. Only those processes and procedures which have the effect of augmenting the total coronary circulation can be effective when there is a progressive and severe diminution in the total myocardial flow.

Such possible augmenting processes include the overdevelopment of normal intracardiac openings which might permit direct passage of blood from the chambers into the deeper myocardial sinuses (sinusoids), and the development of additional vascularity at the base of the heart, and in adhesions occurring between the heart surface and the adjacent pericardium (extracardiac circulation). Such natural compensatory processes may, in certain unusual individuals, entirely replace the normal coronary arterial flow when the latter gradually becomes completely obliterated.[7] However, these natural processes are relatively inadequate to protect completely the average affected individual from the effects of a sudden major coronary occlusion, and so cannot be relied upon.

Attempts have been made by Beck,[8] O'Shaughnessy,[9] and others to augment the effect of these natural mechanisms by suturing adjacent vascular tissues (omentum, lung, pericardium, pectoral muscle) to the surface of the heart. Beck has made a point of removing the epicardium with a burr in order to eliminate this possible anatomic barrier to adequate vascular communion between the graft and myocardium.

Thompson and Raisbeck[10] have used powdered talc in order to produce vascularized adhesions between the pericardium and the epicardium. Beck and co-workers,[11] after extensive animal experimentation, suggested the use of powdered asbestos which they considered more effective than talc. Asbestos not only stimulates the production of vascularized adhesions between the epicardium and the pericardium, but leads to an extensive dilatation and overdevelopment of normal intercoronary communications. It is interesting that some patients so treated seem to have been improved.

It is our considered opinion that only a very small percentage of the normal coronary flow can be replaced by such extracardiac sources. Thus

these measures really afford only partial protection against immediate death from a sudden arterial occlusion, and a certain measure of possible prolongation of life and comfort. For these reasons, Beck[2] has inevitably turned his attention to the possibilities of largely or completely replacing the normal coronary blood flow. He hopes thus to permit a complete or appreciable restoration of the patient to health and activity. He has experimentally supplemented the coronary arterial flow by shunting arterial blood through the coronary sinus into its tributaries.

There are those who question the possibility of retrograde blood flow through the coronary sinus system into the myocardial capillary bed. We propose to show that this flow can be reversed when arterial blood is shunted into the coronary sinus, at least after subsequent narrowing of the sinus near its exit into the right atrium has been accomplished.

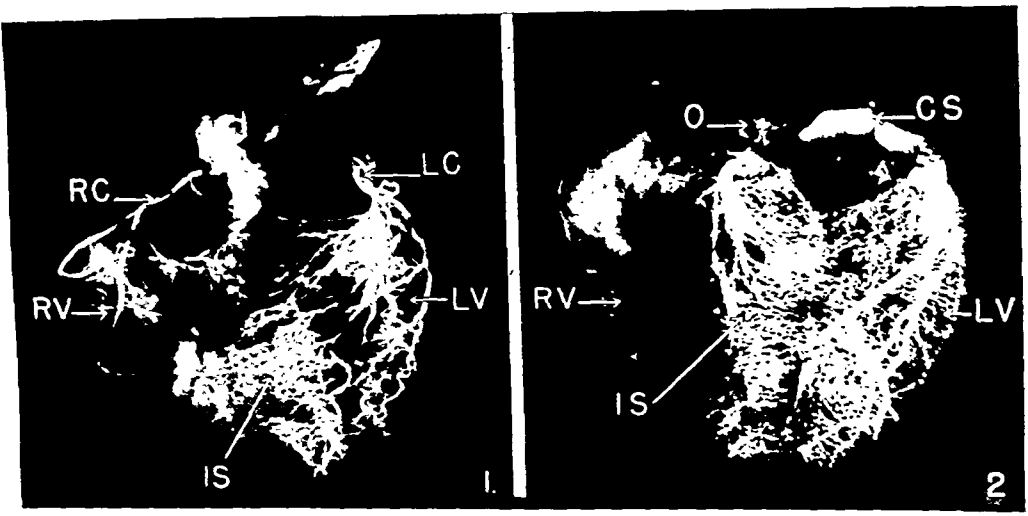
Others[12] have intimated that natural communications exist between the superficial cardiac and the deeper myocardial veins which drain directly through small openings into certain of the cardiac chambers. Such communications would permit arterial blood entering the coronary sinus to escape by way of the myocardial veins. Such blood would be lost from the capillary bed and hence could not nourish the cardiac muscle. We are aware that such communications exist[13] and feel that they afford a valuable safety mechanism regulating the retrograde flow. They do not defeat the physiologic purpose of the operation. While some of the blood admittedly escapes through them into the right atrium, a residual quantity may be distributed through the tributaries of the coronary sinus into the capillary bed of the myocardium. The amount of this capillary flow may be proportional to the deficiency in supply from the coronary arteries. Should too great a quantity of blood enter the sinus by virtue of the establishment of a very large graft, these communicating veins will undoubtedly permit the excess to drain off into the cardiac chambers. Thus, engorgement and damage to the myocardial capillary bed are avoided. Furthermore, these communications, especially those involving the epicardial veins, represent the only possible route for getting blood from the coronary sinus to the right ventricular myocardium.

ANATOMY

Recent anatomic studies by one of us (R. C. T.) have revealed some information on the coronary circulation which is of considerable significance in the Beck procedure.[14] By means of wax model reconstructions of the human newborn heart, it was possible to demonstrate continuity between the large veins and venous sinuses of the ventricular myocardium and the lumen of the coronary sinus. Similar continuity was demonstrated between the myocardial venous channels, the lumen of the right atrium, and the cardiac veins lying in the epicardium. It is interesting to note that many of these thin-walled venous sinuses were found to accompany and to ramify in the common bundle of conduction tissue as they crossed the fibrous ring of the heart.

These microscopic observations on the venous supply of the heart have more recently been verified and extended by the usage of injection experi-

ments, radiographic films, and study of serial celloidin sections (Angulo and Truex[15]). These authors studied both the arterial and venous systems of the human and dog heart with a standardized barium sulfate-gelatin solution. By comparing the arterial and venous injected specimens they clearly demonstrated that, in the adult, the left ventricle and interventricular septum possesses predominant arterial and venous systems, whereas the right ventricle has a more meager vascular supply (Fig. 2, *A* and *B*). Greater significance is attached to their discovery that, within the left heart, the venous tributaries of the coronary sinus form an extensive network which is several times more abundant than that formed by corresponding branches of the left coronary artery.[13] This rich venous plexus, formed by tributaries of the coronary sinus, has been found to be continuous distally with the myocardial capillary bed.[16]



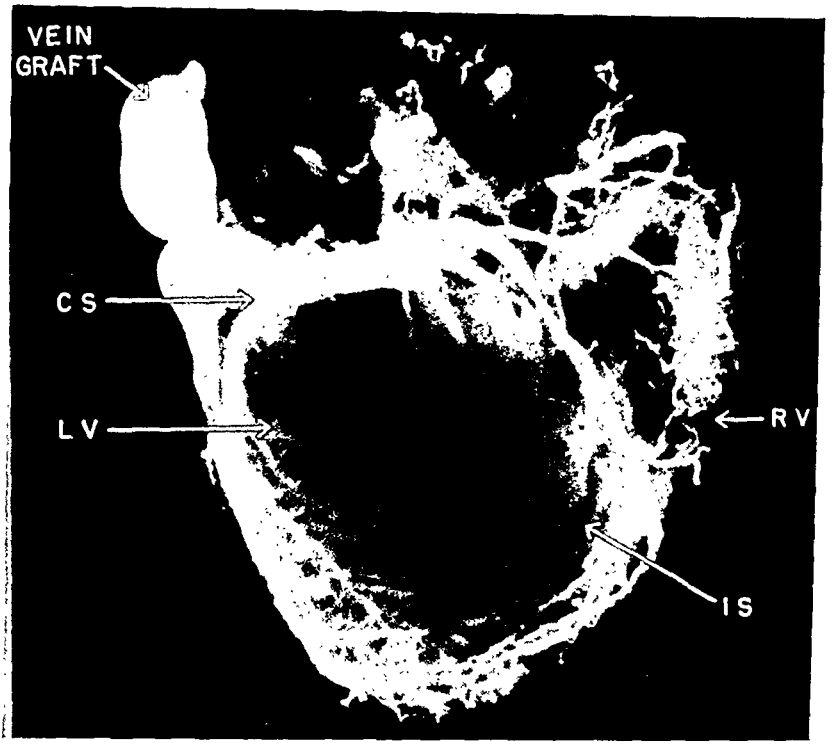
A. *B.*

Fig. 2.—*A*, Injection study of a human heart through the coronary arteries. *B*, Injection study of a human heart through the coronary sinus system. This reveals the numerous venous channels throughout the left ventricle and the interventricular septum. Note the small amount of filling over the right ventricle. (Courtesy of Wistar Institute.)

Hence, this anatomic work has indicated that when the normal arterial system of the heart becomes partially obliterated, as in atherosclerosis, the venous tributaries of the coronary sinus can by surgical intervention provide an alternate route to the capillary bed to meet the functional demands of the left ventricle and septum.

Injection studies have been completed on three dog hearts following the Beck procedure. All of these specimens demonstrate that following their arterialization, the branches of the coronary sinus establish large epicardial anastomoses with the small cardiac and anterior cardiac veins of the right ventricle. Through these anastomoses the right ventricle may also receive arterial blood after the Beck procedure (Fig. 3, *A* and *B*).

The vascular connections of the coronary sinus have also been studied.[13] It possesses anastomoses with the cardiac veins of the right ventricle; myo-



A.



B.

Fig. 3.—*A*, Injection study of a dog's heart following a Beck procedure. The injection media has been introduced by way of the vein graft into the coronary sinus. *B*, The same heart, opened after the method of Schlesinger. Note the vascular channels demonstrated throughout the heart and especially those of the right ventricle, due to the opening up of epicardial anastomoses by the pressure-head of blood entering the coronary sinus by way of the aorta. Compare with Fig. 2, *B*.

cardial veins and venous sinuses of the atrium, interventricular septum, and the left ventricle; and with the lumen of the right atrium. These collateral channels of the coronary sinus serve as a safety valve mechanism after the Beck procedure. Such collateral channels also return the major outflow to the lumen of the right atrium as indicated by their word schema (Fig. 4).

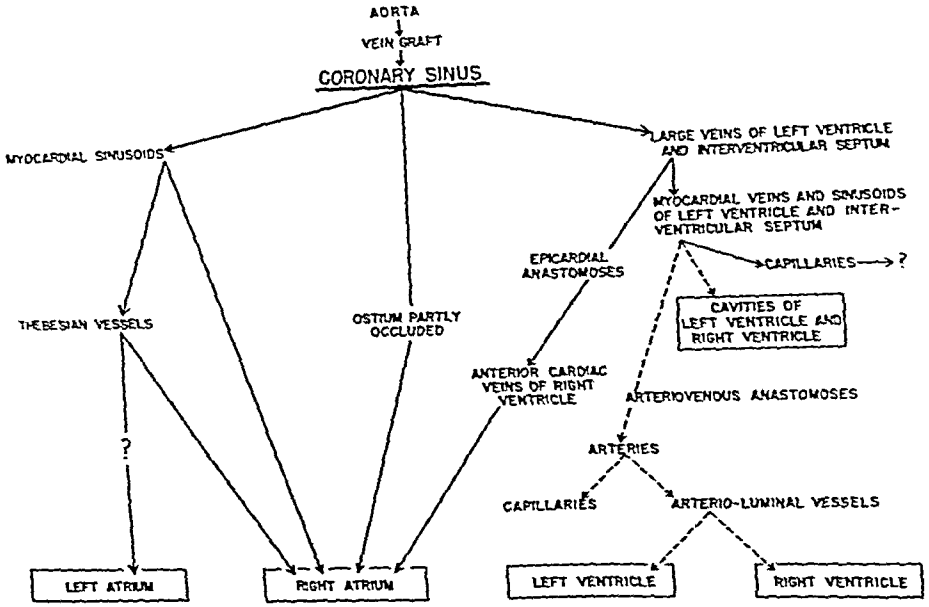


Fig. 4—Schematic representation of the pathway of blood entering the heart following a Beck procedure. The solid lines represent pathways which we have demonstrated. The broken lines represent pathways which are either theoretical or have been reported by others but which have not been demonstrated by us.

RESULTS OF ANATOMIC INVESTIGATIONS

Radiologic films and microscopic methods all demonstrate that:

1. The left heart and septum possess a much richer arterial and venous system than the right heart.

2. In the left ventricle and septum the venous system is far more abundant and extensive than the arterial system. This extensive venous network is drained by tributaries of the coronary sinus.

3. The coronary sinus and its tributaries possess continuity with the capillary bed of the left ventricle and septum. The coronary sinus also has several collateral channels leading to the lumen of the right atrium.

4. Arterialization of the coronary sinus by the Beck procedure permits arterial blood to enter the capillary bed of the left heart by retrograde flow through tributaries of the coronary sinus.

5. Following its arterialization, branches of the coronary sinus establish epicardial anastomoses with the small cardiac and anterior cardiac veins of the right ventricle. Through these anastomoses, the right ventricle also receives arterial blood after the Beck procedure.

6. Collateral venous connections of the coronary sinus serve as a safety valve mechanism after the Beck procedure. These collateral channels also return the major outflow to the lumen of the right atrium.

ANATOMIC CONCLUSIONS

Anatomic findings, to date, indicate that the Beck procedure offers vascular benefits to the entire heart in cases of advanced coronary arteriosclerosis. Better nourishment of the myocardium is attained by a combination of the following two factors:

1. Retrograde flow of arterial blood through the extensive venous tributaries of the coronary sinus provides new avenues to the capillary bed as well as drainage routes back to the heart chambers.

2. Development of anastomoses between branches of the coronary vessels and extracardiac arteries occurs at the base of the heart with the arteries of the pericardium, vasa vasorum of the aorta and pulmonary vessels, and new arterioles which grow into the wall of the vein graft.

PHYSIOLOGY

Physiologic studies in animals or patients upon whom arterialization of the coronary sinus has been performed are extremely few. Eckstein,[17] so far, seems to be the chief investigator to consider this problem.

In a series of dogs subjected to division of the left circumflex coronary artery before, and at varying periods of time subsequent to, performance of the first and second stages of the Beck operation, Eckstein was able to show dramatic changes in the amount and type of coronary flow from the cut end of the distal segment. The average changes are illustrated in Chart 1.*

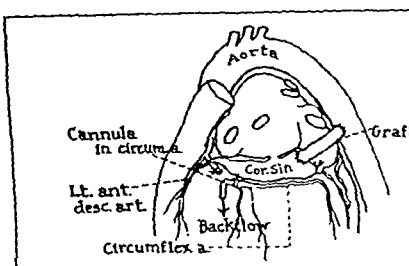
To simplify these findings, we may say that in the normal dog the blood flow from the cut end of the distal segment of the divided left circumflex coronary artery will average 2.5 c.c. of arterial blood per minute. This is presumably derived from the normally existing minute intercoronary anastomoses which bring blood from the remaining intact coronary trunks to the severed one. When a vein graft is anastomosed between the coronary sinus and the aorta, and the sinus outlet is largely obstructed, this flow from the cut coronary artery rises to 20 or 30 c.c. per minute. But the blood is dark and essentially venous, indicating that it has passed through a capillary bed. Presumably it is derived from the arterial blood which has traversed the coronary sinus, its ramifications, and the capillaries by retrograde flow.

With the passage of time this blood from the distal cut coronary end becomes more and more arterial in nature until after twelve weeks it is fully saturated with oxygen. It is thought that this phenomenon is due to progressive enlargement in the intercoronary communications until they are able to replace fully the interrupted normal coronary arterial flow. Undoubtedly the development of such efficient collaterals would be slower and less reliable in human patients with presumably generally diminished flow in all major coronary arteries.

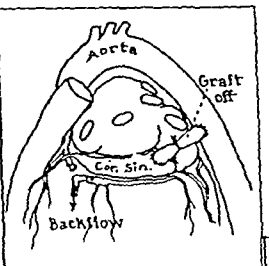
*This chart is used through the courtesy of R. Eckstein and D. Leighninger of Cleveland, Ohio. (To be published.)

Having established what we believe to be a sound, theoretical, anatomic, and physiologic basis for arterialization of the coronary sinus, it now remains to show its surgical or technical feasibility and its clinical effectiveness.

We are indebted to Dr. Claude Beck who, having developed this surgical procedure almost singlehandedly, freely offered us not only the fruits of his fifteen years of animal experimentation, but also the advantages of his personal participation and surgical skills. We were thus able to start a clinical program of surgery for coronary artery disease at Hahnemann Hospital in Philadelphia, Pa., and at St. Michael's Hospital* at Newark, N. J. Rigid



GRAFT OPEN



GRAFT OFF

TIME	BACKFLOW cc/min.	BACKFLOW cc/min.	DIFFERENCE IN OXYGEN CONTENT Vol.%
Normal dogs without arterialization (at time of placing graft sinus press. at 50 mm. Hg.)	7.6	2.8	10.9
At time of sinus ligation (3 wks. after placing graft)	20.8	5.6	8.2
1 week after sinus ligation	22.0	10.0	9.4
2 weeks	23.0	8.0	9.9
3 weeks	24.0	19.0	3.6
1 month	30.0	28.0	2.1
3 months	16.0	20.0	0.0
4 months	11.0	15.0	0.0
6 months	20.0	22.0	0.0

Chart 1.

requirements for the selection of patients for surgery were set up. As a result of this selectivity we were able to show an operative mortality up to the present time of only two cases of the eighteen patients operated upon. This record clearly proves that patients with serious impairment of coronary flow can withstand these operative procedures if the disease process is not too advanced. The operation is not an intracardiac one but merely a meticulous anastomosis performed between a systemic artery, the aorta, and the coronary sinus. This does not cause any severe physiologic disturbance of cardiac function even during the actual performance of the surgery. Therefore, there is no reason why the inherent operative mortality should be high.

*The patients operated upon at St. Michael's Hospital are included with the permission of Dr. Anthony Crecca, Chief of Thoracic Surgery.

SELECTION OF PATIENTS

Criteria for the selection of patients have been established. However, the present arbitrary standards undoubtedly will be modified by further clinical experience.

Disability resulting from atherosclerotic disease of the coronary arteries varies. It is imperative that the operation be performed only for a degree of incapacity that warrants major surgical interference. At the same time the operation should not be considered where the disease is so advanced that benefit or survival cannot be reasonably expected.

For this reason certain indications and contraindications as to operability have been used for the selection of patients. In the short medical history of coronary artery disease many facts have emerged as important prognostic guides. These have been utilized as the basis for further classifying the indications and contraindications as either major or minor.

Experience with the selection of cases for mitral commissurotomy has proved that clinical facts must be evaluated in the light of all attending circumstances. Classification schemes for operability are too inelastic to be practical. Few patients are entirely acceptable or unacceptable for surgery. Proper selection depends upon the critical appraisal of the indications and contraindications and an analysis of their respective importance in a particular patient.

Indications.—The basic requirement for surgery is major or progressive atherosclerotic disease of the coronary arteries. The diagnosis must be established beyond question and confirmed by definite abnormality in the electrocardiogram.

The functional impairment of the patient is the most important consideration. Anginal distress must be frequent and severe enough to interfere seriously with ordinary activity before it can be classified as major. Angina decubitus may be a particularly important indication. In all instances a reasonable trial with the usual medical routines should precede surgical treatment.

The presence of a previous myocardial infarction lends significance to the effort syndrome and increases the advisability of surgery. Where a myocardial infarction has not taken place, the most reliable history of anginal pain must be supported by proper abnormality in the electrocardiogram before it can be considered a major indication. Effort pain, however significant, without electrocardiographic abnormality does not establish the diagnosis of coronary artery disease beyond question, at least in relation to the surgical treatment.

The uncertainty of coronary artery disease, with its implied threat of sudden death, cannot be considered alone as the primary reason for operation at the present time. The basic requirement of disabling angina must be supported by evidence of a previous myocardial infarction or definite electrocardiographic abnormality before it becomes a major indication for surgery.

At this time it appears that patients who have electrocardiographic evidence of myocardial damage due to coronary artery disease, but who are not incapacitated, should not be considered as candidates for surgery even though they have had a previous myocardial infarction.

The significance of symptomatic coronary artery insufficiency is altered considerably by the age of the patient. The presence of the disease before the age of 40 is a major indication for operation. This probably holds true through the fourth decade. From that point, age ceases to become a major indication. Indeed, after 55 it acts as a deterrent.

Normal blood pressure and normal heart size increase the importance of any major considerations for surgery.

The various abnormalities which may appear in the electrocardiogram cannot be rigidly classified according to their importance. Those changes which appear constantly in the tracing probably represent more extensive pathology than those appearing after exercise. However, they may not represent a more significant prognostic guide.

Contraindications.—A recent myocardial infarction is a major contraindication. The operation should not be performed until six months have elapsed. Care must be taken that a severe, easily initiated effort syndrome does not indicate a developing infarction. When serial electrocardiograms show rapidly progressive myocardial damage, the operation must be deferred until the pattern has become fixed for four to six months. This applies even though there may have been no change in the patient's symptoms.

Two myocardial infarctions within one year is a major contraindication. This probably holds true for all patients with multiple infarctions regardless of the time interval.

Blood pressure values which remain above 180 mm. Hg systolic and 100 mm. Hg diastolic are considered firm contraindications. Evanescent readings beyond these levels are of lesser significance at this time.

Cardiac enlargement up to 10 per cent of predicted values is a minor deterrent. As the size increases thereafter this factor becomes of major importance.

The presence of congestive heart failure is an absolute contraindication. This is equally true when gallop rhythm is present. Heart failure that has responded to medical treatment still remains a major contraindication. It would appear that the degree of myocardial damage usually found in patients who have suffered congestive failure on the basis of coronary artery disease is too extensive to be aided by operative interference. However, undue cardiac enlargement, prohibitive hypertension, or multiple infarctions are more likely to be found in this group.

Certain associated diseases may serve as minor contraindications. These would include emphysema, bronchiectasis, diabetes, and marked obesity. Renal failure obviously would gravely compromise the surgical risk.

For technical reasons a markedly calcified aorta must be accepted as a distinct contraindication.

At the present time any single major contraindication is sufficient reason to deny surgery. Such issues as recent or multiple infarctions, congestive failure, gallop rhythm, gross cardiac enlargement, or even moderate hypertension cannot be counterbalanced by any major indication for operation. These patients are the least likely to survive or, if they do, the least likely to benefit.

The ideal candidate offers no major or even minor contraindications. The acceptable candidate may offer one or two minor deterrents which are of limited significance when compared with the major indications that are present.

Preoperative Management.—Patients who have been selected with care for surgery rarely present difficult problems in preoperative management.

A careful history will reveal the circumstances under which anginal seizures occur. Every attempt should be made to avoid duplicating these situations during hospitalization. Where necessary activity is restricted to a minimum, emotional tension is likewise reduced as much as possible. In this regard the period of laboratory and clinical investigation should be regulated according to the patient's tolerance and not to an inviolate routine. Brief words of explanation often sustain a patient through the most trying type of study.

Nitroglycerin is an invaluable drug, and there is no contraindication to its use in the preoperative period. Occasionally the so-called coronary vasodilators may aid in the control of frequent cardiac pain. Rest and mild sedation, however, appear to be the most effective measures before surgery.

Major arrhythmias are unusual in this group of patients. Premature ventricular systoles and the implied threat of ventricular fibrillation are best controlled by oral quinidine or Pronestyl Hydrochloride. These drugs are discontinued at least twenty-four hours before surgery since they may potentiate the effect of the intravenous procaine which is used routinely during the operative procedure.

There is no purpose in digitalization prior to surgery. Indeed, the drug may be harmful in initiating troublesome arrhythmias or conduction defects. Where this drug has been given in the past it should be discontinued unless a pressing indication for its use is still present.

Postoperative Management.—The major cardiac sequellae which develop after the first stage procedure are a persistent sinus tachycardia, premature atrial contractions, or atrial fibrillation. The sinus tachycardia requires no specific therapy. It does not respond to digitalis and usually subsides on the fourth or fifth postoperative day. Frequent premature atrial contractions usually precede atrial fibrillation. The latter, however, has occurred with no previous indication of atrial irritability. When the ventricular response to atrial fibrillation is uncommonly rapid, and the integrity of the circulation threatened, or when congestive failure actually develops, digitalis or any of the related compounds may be given. If failure is not present, however, the drug should be withheld if possible. The rhythm will revert spontaneously during the first week. Occasionally quinidine has been given under these circumstances but the effect is questionable. One patient (A. D.) continued to show evidence of atrial irritability for two weeks after both the first and second stages even though full doses of quinidine were given.

Oxygen is a routine medication for the first twenty-four hours. It is rarely required for a longer period of time. The patient is encouraged to cough from the start. Retained secretions are promptly treated with aspira-

tion, bronchoscopy, or tracheotomy as indicated. The patients are fed a soft diet within twenty-four hours and are encouraged to feed themselves as soon as is feasible thereafter. This applies to either stage of the operation.

Attention to bowel function is usually deferred until the third day when a warm saline enema may be given if necessary.

Ambulation depends upon the febrile reaction, cardiac rate, and the general condition of the patient. Generally it is not attempted until the seventh to tenth postoperative day. Frequent leg movements in bed can alleviate the discomfort and inherent danger of too early activity in the cardiac patient. When walking is finally permitted, it is regulated to the patient's tolerance and not to a set routine.

Antibiotics are routinely used. Penicillin and terramycin have been favored. There is, however, no need for anticoagulant therapy. Patency of the graft apparently depends upon technical factors and not upon the incoagulability of the blood. The routine use of aminophyllin by vein may have a beneficial effect upon the coronary circulation without greatly increasing cardiac output or the danger of arrythmia.

Immediately after the first stage operation a to-and-fro bruit may be heard at the second left interspace anteriorly or high in the interscapular area to the left of the spine. Even though the graft remains patent, this murmur may become considerably diminished on the third or fourth day. It then generally reappears and remains constant. Tilting the patient to the right at times increases the intensity of the sound. It is generally best heard after a full expiration. No other murmur develops as the result of the surgery.

The postoperative period is usually well tolerated. Patients have shown no reluctance to enter the second stage after successfully withstanding the discomforts of the first.

TECHNIQUE

First Stage.—The original Beck technique as applied to human beings is as follows: A segment of a large superficial vein, preferably without valves, is removed under local anesthesia. Either the median basilic vein of the arm or the external jugular vein is usually chosen. If a valve-bearing segment cannot be avoided, the vessel must be marked so that the proposed flow of blood will be in the direction of the valve. All branches to the vein must be carefully ligated, and any excess adventitia or perivascular tissue is carefully dissected away. The segment is irrigated with heparin solution and placed between pads of moist gauze until needed.

Preanesthetic preparation,* medication, and anesthesiologic care are directed toward (1) decreasing the irritability of the heart, and (2) lessening any factors such as nausea, coughing, or straining that may be present just before or during operation or in the immediate postoperative period.

Preliminary medication is aimed toward having the patient rested, and the metabolic rate as nearly normal as possible without depression. Pentobarbital sodium (Nembutal), 0.10 to 0.2 Gm. (1½ to 3 gr.), or seco-barbital

*The anesthesia for these patients has been managed by Dr. Kenneth Keown of the Department of Anesthesiology at Hahnemann Hospital.

(Seconal Sodium) in the same dosage is prescribed the night before operation. From an hour to an hour and one-half before anesthesia, 0.1 Gm. of pentobarbital sodium (1½ gr.) is given by mouth, and meperidine hydrochloride (Demerol) 75 to 100 mg., with atropine sulfate, 0.60 mg. (1/100 gr.) are given intramuscularly. Shortly after administration of these drugs, a vein in the forearm or wrist of the right arm is cannulated under infiltration anesthesia.

After the patient is brought to the operating room, absolute quiet is essential. The equipment for making electrocardiographic tracings and determinations of blood pressure is applied after the patient has been placed in a supine position on the operating room table.

Ten minutes before induction of anesthesia, a solution of 0.2 per cent procaine in 5 per cent dextrose and distilled water is administered intravenously in the operating room. From 200 to 300 mg. of procaine is given before tracheal intubation is carried out.

Thiopental sodium (Pentothal Sodium) is then given intravenously in dosages sufficient to produce hypnosis (150 to 200 mg.), followed immediately by the intravenous injection of decamethonium bromide (Syncurine), 3 to 4 mg., and then 150 to 200 mg. more of thiopental sodium (Pentothal Sodium). By this time approximately 350 mg. or more of procaine has been given. A Magill endotracheal catheter with cuff is passed under direct vision by the oral route. A mixture of 50 per cent nitrous oxide and 50 per cent oxygen is administered from the time the patient loses consciousness.

During all steps apnea is avoided with its ensuing, although temporary, hypoxia. Respiratory function is augmented or controlled to insure adequate pulmonary ventilation with both adequate oxygen delivery and carbon dioxide elimination. The Beck-Rand respirator is most useful in these cases. Administration of nitrous oxide and oxygen in this concentration is continued throughout the course of anesthesia.

Procaine is continued at the same concentration of 0.2 per cent, with a basic flow of 1 Gm. per hour. Thiopental sodium is added as required for basal hypnosis. We believe analgesia is afforded by intravenous procaine.

At the completion of the anastomosis, the rate of flow of procaine is decreased. Usually thiopental sodium need not be added after the approximation of the chest wall has been started. We attempt to have these patients awake, moving, and responsive at the completion of surgery.

The usual curved posterolateral thoracotomy incision is placed over the left seventh interspace, being extended as far anteriorly as the midclavicular line. Bleeding points are ligated with fine silk. The seventh rib may be removed subperiosteally if added exposure is desired. Large fixed retractors are used to obtain exposure. The lung, if adherent, is detached from the chest wall, diaphragm, and mediastinum by blunt and sharp dissection, and the inferior pulmonary ligament is sectioned up to the inferior pulmonary vein. The lung is thereafter kept retracted upward by large warm gauze pads with or without the use of hand-held retractors. The diaphragm, if high or tending to move, is held down by gauze-covered, hand-held retractors.

The pericardium is opened from a point 3 cm. from the ventricular apex to the middle of the left atrioventricular groove, and then extended from this point to expose the left atrium and the termination of the inferior vena cava. The heart surface is inspected for evidences of previous infarction and the major coronary arteries are examined for sclerotic changes. All cut pericardial edges are supported by application of traction sutures at 2 cm. intervals (Fig. 5).

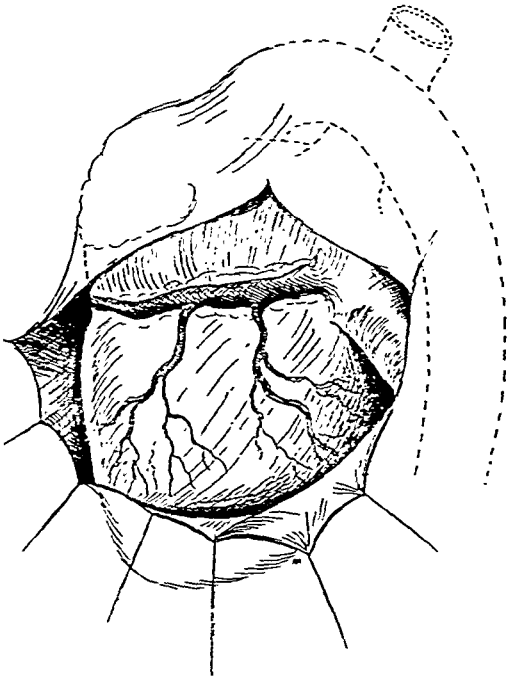


Fig. 5.

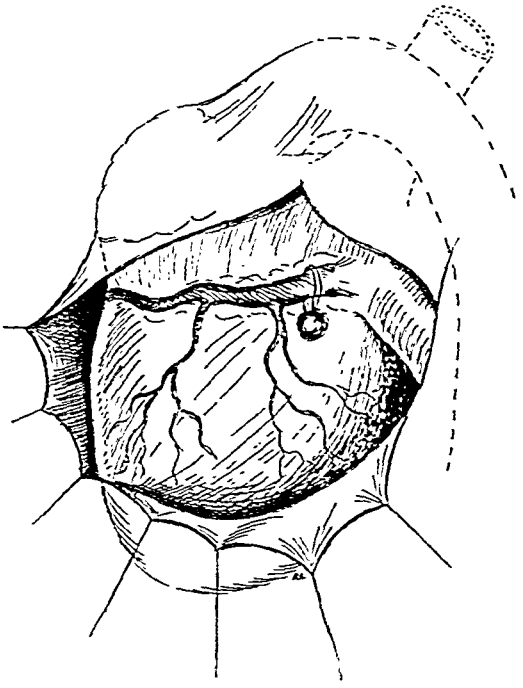


Fig. 6.

Fig. 5.—Exposure of the coronary sinus through the open pericardium.
Fig. 6.—The Orlon sutures are shown encircling the coronary sinus, ready for subsequent sinus ligation at the second stage.

The coronary sinus is identified and is traced to its terminus into the right atrium. It is partially dissected free from the epicardial fat in which it is somewhat imbedded, just above the point of termination. Traction sutures applied in the anterior lip of this epicardial fat may render the subsequent maneuvers easier. A curved half circle, one-inch needle is now threaded with a heavy plastic (Orlon) suture. The needle is passed completely around the sinus about 1 cm. from its terminus. Great care is taken that the needle does not puncture the sinus. The appearance of dark (unoxygenated) blood through the needle wounds is indicative of such puncturing, and requires that the suture be removed and the needle repassed. Actual passage of the needle through or around some of the muscle fibers of the atrial wall is acceptable.

This double suture is to be tied down at the second operative stage to obstruct partially (largely) the exitus of blood from the coronary sinus into

the right atrium. This step will ultimately insure the reversal of flow through the coronary sinus and its ramifications.

The needle is removed, and the ends of the sutures are matched up and tied together for subsequent ease of identification. They are then rolled upon a fine pair of hemostats, and a 00000 (arterial) black silk suture is passed through and around the tiny coil to maintain this compact and untangled state. The coil is then sutured out of the way under the heart and within the pericardium for easy accessibility and identification at the second operative stage, at which time reactive processes and adhesions will have largely obliterated identifying landmarks (Fig. 6).

A suitable site in the length of the coronary sinus is now selected for the anastomosis. This may be at any convenient point proximal to the previously applied Orlon suture. The apparent caliber of the sinus, its prominence and ease of mobilization, and its proximity to the thoracic aorta are all considered in the selection of the anastomotic site. Again the adjacent or imbedding fat is partially dissected away on either side of the sinus. Traction sutures may be used in the inferior (anterior) lip of fat.

Having partially mobilized the sinus in this region, two 00000 arterial silk sutures swedged upon atraumatic curved needles are passed longitudinally into the most prominent aspect of the sinus wall, taking bites about 3 mm. long, parallel to, and about 3 mm. apart from each other. Bleeding should be free from each needle puncture, indicating that the lumen of the sinus has been fully penetrated. If such bleeding is absent, the suture should be removed and replaced to prevent subsequent separation of the intima since this will cause difficulty in entering the sinus for performance of the anastomosis. Slight traction upon the stay sutures facilitates inclusion of a satisfactory amount of sinus wall for exteriorization within the jaws of the special curved sinus clamp (Fig. 7).

A No. 11 Bard-Parker bistoury-type scalpel blade (on an appropriate handle) is now used to make a longitudinal puncture opening in the excluded portion of the sinus midway between the two stay sutures (Fig. 8). This opening is extended in length for a distance of 6 or 8 mm. using a pair of angulated iris scissors. The size of the opening depends upon the size of the graft available for the anastomosis. The sinus lumen is then flushed with heparin solution.

A thirty-six-inch 00000 arterial silk suture with an atraumatic curved needle at each end is used to attach the end of the graft to the inferior (caudal) extremity of the incision in the sinus (Fig. 9). The suture is tied so that both ends are of equal length. One end is temporarily placed out of the operator's way while the other end is used to perform the anastomosis of the inferior (anterior) lip of the incision. This is accomplished by a continuous running, suture technique, the needle at each bite passing from the adventitial side of the graft, about 1½ mm. from the free edge, into the graft lumen, then from within the sinus through its wall and out the adventitial side. An attempt is made to place the sutures 1½ mm. apart in the graft. When the suture line reaches the longitudinal stay suture, the latter is removed. After

the suture line is completed, each stitch is individually picked up and tightened by the use of fine Potts type forceps. A separate stay suture may be placed at the upper (cephalic) extremity of the incision and tied to the end of the anastomotic suture. The suture line on the other side of the graft is similarly applied, and the free end is tightened and then tied to the end of the initial suture. Unless there has been puckering of the sinus opening, there is now a very adequate anastomotic communication between graft and sinus. The sinus clamp is removed permitting dark (venous) blood to run into the graft and out of its free extremity. The suture line is inspected

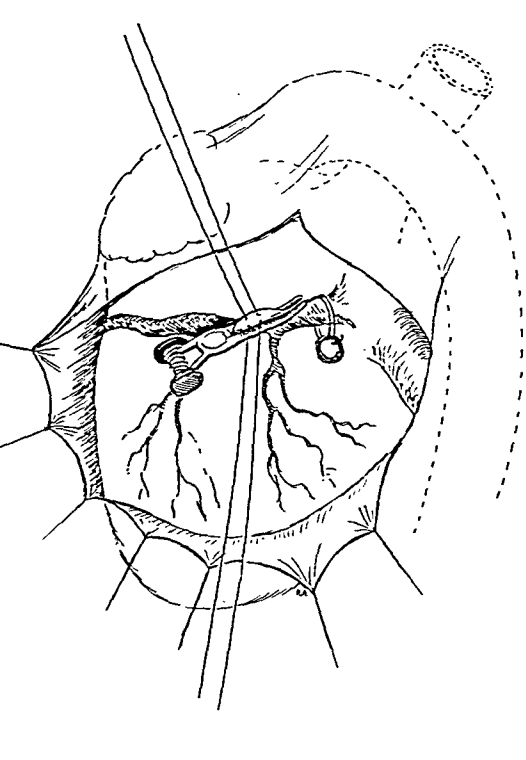


Fig. 7.

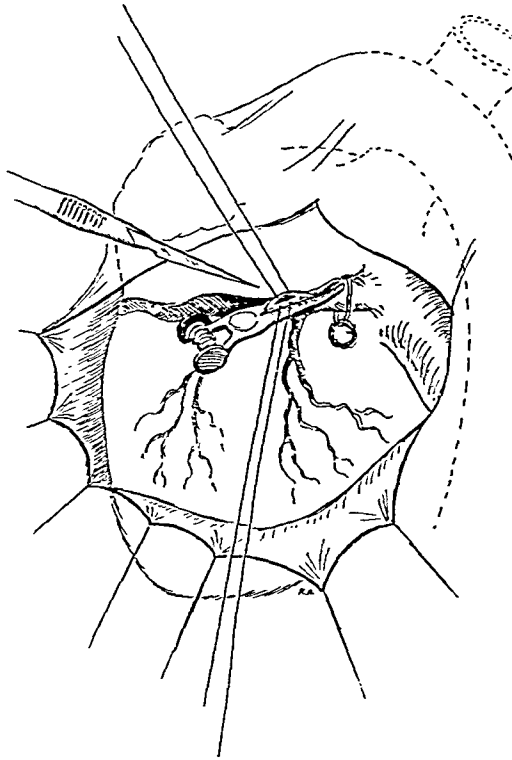


Fig. 8.

Fig. 7.—The site for anastomosis in the coronary sinus is tented by stay sutures, and a segment of the sinus is exteriorized by the clamp.

Fig. 8.—This shows the incision in the exteriorized segment of the coronary sinus.

for leaks and may be repaired by applying individual interrupted stitches if necessary. The graft is finally flushed with saline and heparin solution and is clamped off by an old-type rubber-covered bulldog clamp with a rather weak spring (Fig. 10).

Preparation is now made to anastomose the other end of the graft to the aorta. The graft is shortened to a length which is felt to be just adequate for a good anastomosis at approximately right angles to both the aorta and the sinus. All excess mediastinal fat and lymph nodes lying in the way of the graft are removed to avoid possible pressure upon and adhesions to the graft.

The selected site upon the aortic wall is mobilized. Two mosquito hemostats are applied to the aortic adventitia about 1 cm. apart, one cephalad and one caudad. As traction is made upon them, a special Beck toothed clamp is applied to the aortic wall. This clamp is designed to exclude a sizable portion of the aortic lumen while permitting blood flow beneath it. An attempt is made to exclude 1 cm. of the aortic circumference from the residual aortic lumen (Fig. 11). The mosquito hemostats are then removed and a portion of the adventitia is dissected and cut free from the site of the intended anastomosis.

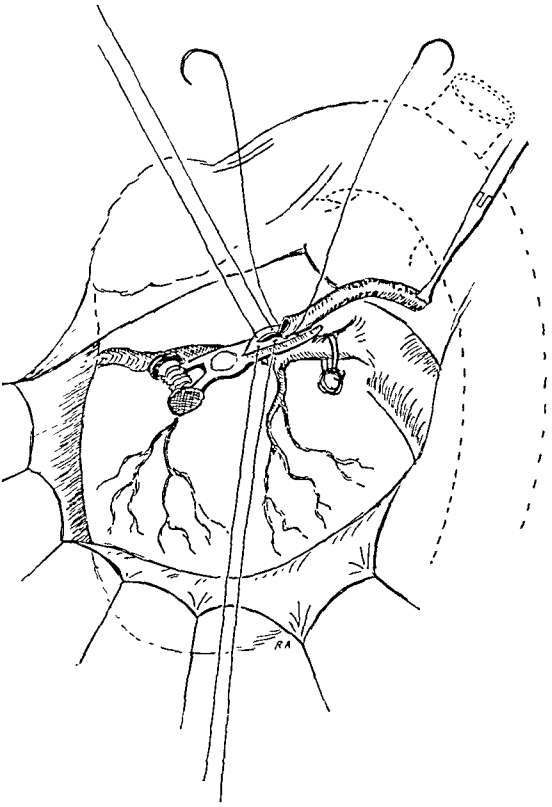


Fig. 9.

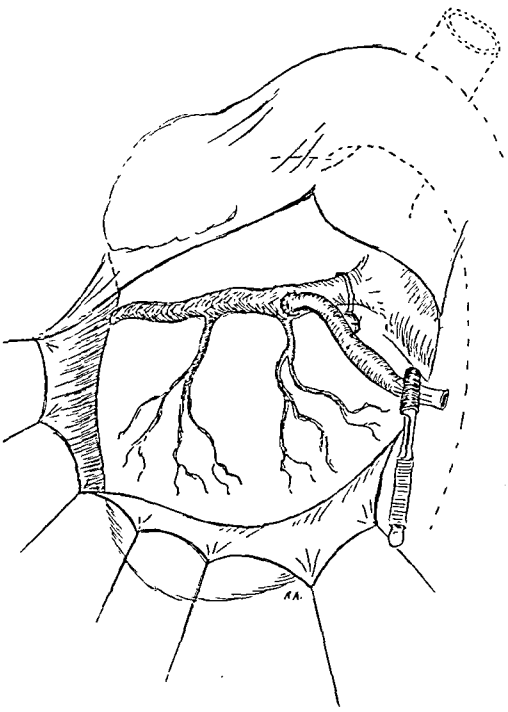


Fig. 10.

Fig. 9.—The beginning of anastomosis between the vein graft and the coronary sinus.
Fig. 10.—The vein graft is anastomosed to the coronary sinus, and the distal end is occluded by the small bulldog clamp.

Using a No. 11 Bard-Parker knife blade and handle, a 4 to 6 mm. longitudinal incision is made into the excluded portion of the aortic lumen (Fig. 12). Then using an identical technique, the free end of the vein graft is carefully anastomosed to the aortic incision. When it is completed, the bulldog clamp is removed from the graft and the suture line is examined for leaks. They may be controlled by individual arterial sutures, if necessary (Fig. 13).

The aortic clamp is gradually released and the suture line is again inspected. Arterial leaks may require only temporary gauze pressure for control or may require additional stitches. Sometimes the aortic clamp must be

replaced temporarily. After the flow through the graft is established, a vigorous continuous thrill is palpable throughout the graft and in the distal end of the coronary sinus. In persons with extremely impaired coronary arterial flow the red arterial blood from the graft may be seen immediately to extend a variable distance into the large veins draining into the coronary sinus. This effect can be produced or greatly enhanced by simply obstructing the outflow of the coronary sinus by traction upon the encircling Orlon sutures. With such traction all the cardiac veins are transformed in color from a blue to a distinct red.

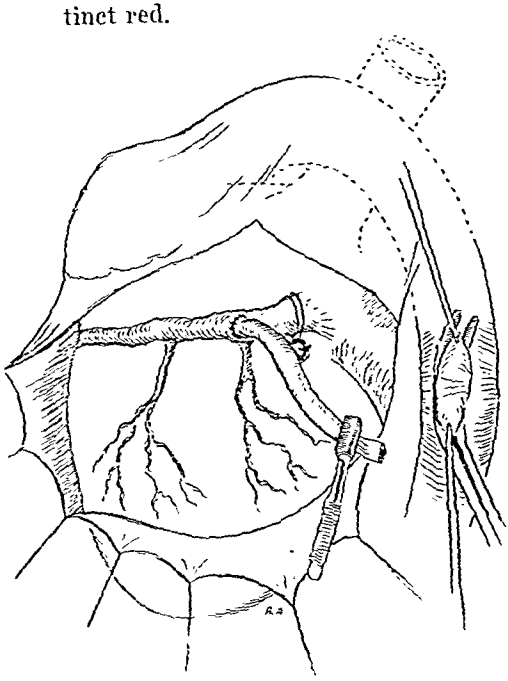


Fig. 11.

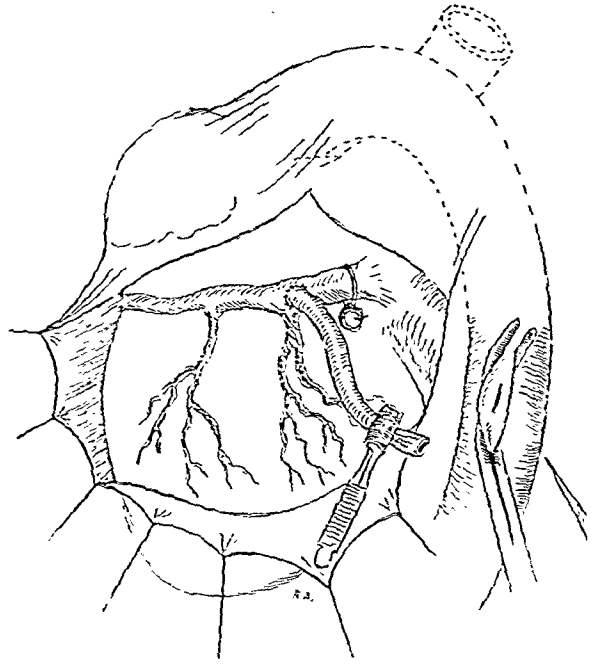


Fig. 12.

Fig. 11.—This shows a segment of the aorta tented by means of mosquito forceps and exteriorized by use of the special clamp.

Fig. 12.—Incision in the exteriorized segment of the aorta.

Second Stage.—Three weeks after the first operative stage, the patient is again anesthetized in much the same manner with an endotracheal airway. He is again positioned laterally, and the former incision is opened.

The lung is usually found to be adherent to the parietal wall, and a variable amount of loculated pleural fluid may be present. The lung is carefully mobilized in the lower thorax exposing the pericardium.

The previous pericardial incision is reopened, and any adherent tissues are carefully dissected away from the region of the graft. A patent graft is easily located by the persistence of the thrill which is found not only in the graft but in the distal portion of the coronary sinus as well.

Then the rolled-up Orlon sutures are located, sutured within the pericardium. They are carefully mobilized and are unrolled by dividing the

black arterial silk. The two Orlon sutures are separated and are then separately tied down over a 3 mm. proble which is laid on the coronary sinus. After tightly tying the suture, the sound is drawn out, thus providing a 3 mm. passageway through which the sinus blood can partly escape into the right atrium. This is repeated with the second Orlon suture (Fig. 14). The chest wound is then closed in the usual manner using a water-seal drainage system. This second stage operation is relatively short.

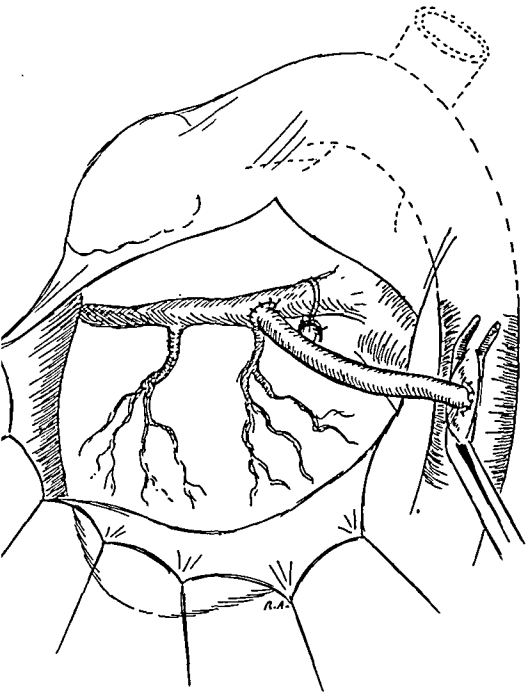


Fig. 13.

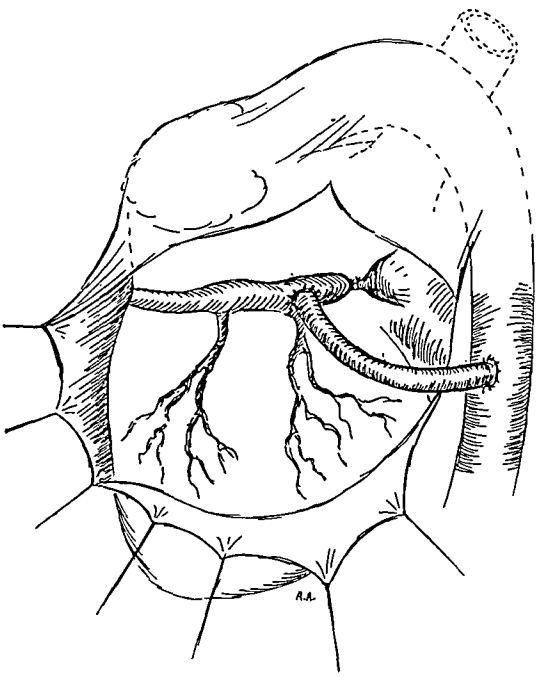


Fig. 14.

Fig. 13.—Completion of the aortic anastomosis and loosening of the aortic clamp for management of any possible leaks at the suture line which might be present.

Fig. 14.—Completed second stage, showing the partial occlusion of the coronary sinus by means of the encircling Orlon sutures.

TECHNICAL MODIFICATIONS

For theoretical and practical reasons (such as the incidence of thrombosis of the graft) we have recently suggested and carried out certain technical modifications of this operation. These have in no respect changed the underlying basic principles.

One of these has been an attempt to use the splenic artery rather than the aorta and a vein graft as the source of arterial blood. While this is technically possible in approximately 75 per cent of patients, it is unsatisfactory in about one-third of these because the terminal lumen of the splenic artery is too small. Thus only about one-half of all patients can expect a good arterialization of the coronary sinus by this means. Such an anastomosis was attempted in two of the present series. In one of them it proved to be impractical because of the small terminal size of the vessel.

Another technical modification, and a much more promising one, is the excision of a button of aortic wall rather than merely a longitudinal incision of the aorta (Fig. 15). Our attention was drawn to this detail when the observation was made during the first stage of one of these operations that the initial heavy flow through the graft became diminished and closed off within a period of fifteen minutes after it was established. This was apparently due to approximation of the lips of the aortic incision shutting off the flow. While this phenomenon has not been observed in experimental animals nor in children subjected to the Potts aortic-pulmonary anastomosis for tetralogy of Fallot, it is quite conceivable that it would not be uncommon in the middle-aged individual with an

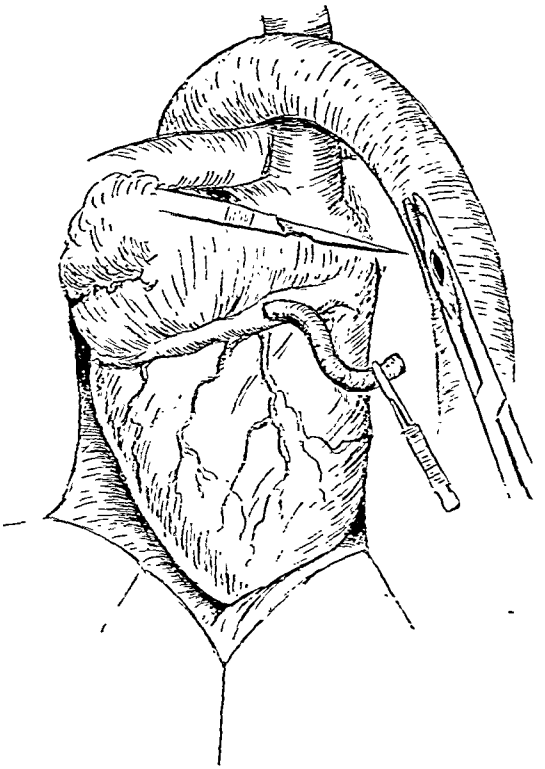


Fig. 15.—Preparation of the exteriorized segment of the aorta by means of resection of a "button" of aortic wall, rather than a linear incision.

arteriosclerotic aorta who is the usual candidate for arterialization of the coronary sinus. Such reduced or actually occluded flow in the graft would almost certainly lead to subsequent thrombosis. In two subsequent cases a button of aortic wall was removed. An unthrombosed graft was the result in each. Use of a free segment of the left subclavian artery, easily available at surgery, seems to provide a superior graft.

Another technical modification which we have carried out in one patient is the use of a continuous everting mattress suture for the aortic anastomosis. While both the vein graft and the coronary sinus have an inherent tendency

to eversion of the cut edge, thus rendering a simple continuous suture line very satisfactory for the anastomosis of vein to sinus, the cut aortic edge has no such tendency. Indeed, the aortic wall tends to separate into layers with the intima detached from the media. It is our feeling that an everting mattress suture would actually partially apply the vein graft into the aortic opening to seal off any such irregularities.

Finally, we have discontinued the practice of removing the adventitia from the aorta, since this provides a much stronger tissue for the insertion and holding of sutures. In an arteriosclerotic, perhaps excessively friable aorta, the considerable strength of the adventitia would seem well worth preserving. The old theory that retention of vascular adventitia predisposes to thrombosis of a vascular suture line was thoroughly disproved in a comparative experimental study by Jamison and Dugan.[18]

We feel that these modified technical details have been responsible for the somewhat improved results obtained with respect to the patency of the graft as observed at the second stage of the operation.

Recent discussion with Beck[19] regarding these technical modifications, particularly that of resection of a button of the aortic wall prior to performance of the anastomosis, has led to this wholehearted concurrence. In fact, he had early in his experience removed an aortic button in a human being. For some reason that technique was then abandoned, but will probably now be revived.

This report is based upon eighteen patients operated upon at the Hahnemann Hospital of Philadelphia and at the St. Michael's Hospital of Newark, N. J. The majority of these patients were operated upon by Dr. Claude Beck himself, and only the more recent of them have been independently operated upon by one of us (C. P. B.). We are endeavoring in this presentation to analyze the results obtained in this operative series and to indicate the various problems and pitfalls encountered.

Two patients in this series did not actually have an anastomosis performed. In one patient (W. C.) the coronary sinus was so delicate that it would not hold a suture for anastomosis, and was finally closed with difficulty. In the other patient (S. S.) the coronary sinus was found to lie too far to the right of the aorta, making anastomosis technically impossible.

There were two deaths in this series, a mortality of 11.1 per cent. One of these was due to sudden exsanguinating hemorrhage at the site of the aortic anastomosis on the seventh postoperative day. The aortic wall had seemed abnormal in this case at the time of surgery, and anticoagulant therapy (Dicumarol) had therefore been instituted and carried out during the entire postoperative period. Whether the abnormal aortic wall (site of an arteriosclerotic ulcer as seen at autopsy) or the anticoagulant therapy had been responsible for the secondary hemorrhage is debatable. This is the only patient in the entire series treated with anticoagulants. The graft was patent.

The other death occurred a few hours after surgery, apparently due to cardiac or circulatory depression. This patient had had two infarctions prior

to surgery, and presented a definitely enlarged heart. Today, we would exclude such a candidate from operative consideration.

One patient (W. S.) developed a pleural empyema after the first operative stage. Adequate drainage resulted in re-expansion of the lungs and complete obliteration of the pleural space. However, a second stage operation was not performed since this patient refused surgery.

In two patients (A. D. and N. R.) thrombosis of the anastomosis was disclosed at the time of the second operation. In one of these the splenic artery had been anastomosed to the sinus. This was treated by immediate application of a vein graft between the aorta and the coronary sinus. The other was treated by opening the graft and removing the clot. The Orlon sutures were then tied down in each case. No subsequent stages were ever performed. Both patients have manifested slight subjective improvement.

Of the remaining eleven patients, four were found at the second stage to have thrombosed grafts. They were treated by instillation of asbestos powder into the pericardium. One is slightly improved, two are unchanged, and one died six months after surgery of progressive disease.

The seven with patent grafts who had both operative stages have presented consistent evidences of improvement varying from what would seem to be "clinical cure" to moderate amelioration of symptoms.

It was our intention to present objective evidences of clinical improvement in this presentation.

Unfortunately, we find that this is a rather difficult undertaking. In the first place, the follow-up is very short for such a variable disease as coronary atherosclerosis. Our oldest patient is now only fifteen months postoperative.

Second, the only valid objective study method which we have employed throughout the entire series is serial electrocardiography. Unfortunately, ballistocardiography was not employed in the early cases. It is being applied in our later cases and will be extensively used in the future.

ECG.—The postoperative electrocardiographic changes observed thus far in human beings appear to resemble those seen in the experimental animals. These changes consist mainly of negative T waves in either the standard or unipolar leads. The full implication of these changes is not entirely understood. The period of observation has been too short to ascertain the degree and rate of return of these changes and their relation to the clinical state.

Consideration of the usual cardiologic methods of evaluating progress in patients with coronary artery disease reveals that they are all basically subjective.

Thus anginal pain, its severity, and its frequency is essentially subjective. Shortness of breath is also subjective unless it is associated with cyanosis, bronchospasm, or pulmonary edema. None of our patients presented any of these last clinical features.

Similarly, the so-called objective tests of work tolerance whether controlled (treadmill, Master's two-step test) or uncontrolled (climbing stairs,

walking, and working) are strictly limited by the former two features: angina and dyspnea, which halt continued activity.

Obviously radiography offered us nothing since the patients in this series did not have much myocardial enlargement and presented no pulmonary congestion, even preoperatively.

Since we excluded cases with congestive failure, we could not measure any diminution of liver engorgement nor of dependent edema.

Thus our evaluation was limited to essentially subjective evidences of improvement. By these criteria, all seven were considerably improved. Anginal pain was completely abolished in the six who had previously presented it. No nitroglycerin has been used by any of the patients postoperatively. Dyspnea was markedly reduced. Work tolerance was so increased that all have been able to return to their former occupations except one, a physician who is less than three weeks postoperative. Stair climbing ability has increased in each case.

All express a great sense of well-being with improvement in appetite and general outlook. Perhaps part of this euphoria is due to a sense of confidence that potential future coronary arterial occlusion will not produce myocardial infarction.

Our earliest case was a patient (J. C.) who had suffered three distinct myocardial infarctions. He has been converted from a cardiac invalid, who took 100 nitroglycerin tablets per week and was unable to walk fifty feet without pain, to a robust man who can with ease run at top speed completely around a city block.

Such important subjective evidence of improvement leads us to feel that a great fuctional change has taken place in each individual's cardiac action. More absolute indications must await other studies and the passage of much more time.

SUMMARY AND CONCLUSIONS

1. The concept that arterial blood can be delivered to the myocardial capillary bed by reversing the flow through the coronary sinus and its ramifications has been presented. Further anatomic and physiologic research is still necessary. Such studies are now under way (by some of us[16]).

2. These techniques show that the vascularity of the left ventricle and ventricular septum is much greater than that of the right ventricle, and that the venous system is far more abundant and extensive than the arterial.

3. After the Beck operation anastomotic communications between the epicardial veins of the left heart (ramifications of the coronary sinus) and the small cardiac and anterior cardiac veins of the right ventricle become markedly dilated. This suggests that they may offer an additional safety mechanism to permit escape of any excess blood entering the heart through the graft. Such collateral veins suggest the possibility of also improving the circulation of the right ventricle by the arterialization of the graft.

4. Existing physiologic information (Eckstein) bears out the probability of immediate reversal of flow through the superficial cardiac venous drainage after the second stage of the Beck procedure.

5. Both anatomic and physiologic evidence bears out the assumption that arterialization of the sinus in time brings about a progressive and great dilatation of the normally minute existing intercoronary communications. Extracardiac anastomoses between the coronary vessels and adjacent structures also become established and functioning. The relative amount of flow through these channels is still unknown.

6. The original Beck anastomotic technique is presented. Certain modifications have been suggested to lessen the incidence of technical failures. Chief among these is the removal of a button of aortic wall to produce a persistent rapid flow of blood through the graft and sinus. It is believed that thrombosis of the graft will, in the future, be a rarity because of this modification.

7. Criteria for the selection of suitable subjects for the procedure are suggested. Surgical survival figures in our experience indicate that such patients can easily tolerate the operation.

8. Objective evaluation of progress in cases of coronary disease under any form of treatment, medical or surgical, is both difficult and rare. The greatest clinicians manage these patients essentially with purely subjective guidance except for the information supplied by the electrocardiograph. Perhaps the ballistocardiograph or some other study mechanism may in the future offer us truly objective criteria.

9. The electrocardiogram is so changed by the reversal of blood flow through the coronary sinus in both dogs and human beings that it is valueless in measuring improvement or regression in these cases.

10. The theoretical advantages and the subjective improvement in patients who successfully undergo both stages of a Beck procedure are so great that we are encouraged to continue this operative program until time and improved methods of measurement ultimately establish its value.

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DISCUSSION

DR. CLAUDE S. BECK, Cleveland.—I should like to express my appreciation to Dr. Bailey and his group for the work they have done in this development. This operation has required assistance and I hope the work will receive the support of a good many of the surgeons in the audience.

I think I can say positively that the operation protects the heart against occlusion of a major coronary artery. It reduces the incidence of ventricular fibrillation after a major artery has been ligated, and it reduces the infarction or the destruction in the myocardium after the artery has been ligated. These, I believe, are statements of fact. We would like to know how to explain this benefit, and during the past year Dr. Eckstein and Dr. Leighninger have been doing experiments to show the mechanism by which this new circulation is beneficial to the heart. This physiologic study is difficult to carry out. They have been working on the study for one year, and it will require another year of full-time work to complete it.

Several conclusions can be drawn from the work so far. It has been shown first by Hahn in a few experiments, and during the past year by Eckstein and Leighninger and others working with them, that arterial retrograde flow from aorta, through graft and through capillary bed does occur. This blood does give up its oxygen. If the circumflex artery is ligated and then cut distal to the ligature, the blood comes out of the cut artery as venous blood. In order to give up its oxygen this blood from the graft must have gone through the capillary bed. Perhaps I should explain the type of experiment that was done. The graft of vein was placed between sinus and aorta. Three weeks later the sinus was partially ligated at its ostium in the right ventricle so that all the blood would not escape as in an arteriovenous fistula. At variable intervals thereafter the circumflex artery was dissected out, ligated at its origin, and cut distal to the ligature. The distal end was cannulated. Studies in back flow from the cut artery were made with the graft open and with the graft closed. I can only refer to these studies in general terms.

One, it has been shown that back flow through the capillary bed does occur. In the dog with normal coronary arteries it would appear that the quantity of such capillary back flow reaches a peak in several weeks and then tapers off and stops entirely. Whether this occurs in the human patient with coronary artery inflow disease we do not know. Two, it has been shown that the graft stimulates the development of intercoronary arterial anastomoses. Why it does this we do not know. In the normal dog's heart the amount of intercoronary back flow is about 2 to 4 c.c. per minute. That is, if the circumflex is ligated and cut the amount that escapes from the cut artery is only a few cubic centimeters and it comes from the descending coronary artery. It is red blood and it has not gone through the capillary bed. As time goes by and as actual retrograde flow from graft through capillary bed decreases, the intercoronary flow increases so that the retrograde flow may be as high as 22 c.c. per minute at the end of six months.

The third point that these experiments by Eckstein have shown is the importance of a few cubic centimeters of oxygenated blood to ischemic myocardium. As small an

amount as 5 c.c. per minute delivered to the muscle mass supplied by the circumflex artery, and this probably is half of the left ventricle, can prevent fibrillation and death. The importance of this does not seem to be generally realized. A fourth point is beginning to appear. They have done experiments in which the back flow from the severed artery was small and it was the same with the graft open or with the graft closed off. At the same time the electrocardiogram approached normal with the graft open and became abnormal with the graft closed off. Perhaps oxygen transfer can take place without the blood making a complete transit through the capillary bed and out the artery. The vascular bed on the venous side of the capillaries is extremely rich. Perhaps there is some ebb and flow here that can be beneficial without going through the capillaries.

It is fortunate that these studies are being made on the dog, and it is fortunate that we have as good a physiologist as Dr. Eckstein and as good a cardiac surgeon as Dr. Leighninger to do this work.*

There are no similar studies for the human heart. Severe coronary artery disease in the human heart produces circulatory conditions that cannot be duplicated in the dog heart. In the human heart with extensive and marked arterial occlusion one would expect the inflow to be greatly reduced and the capillary pressure to be greatly reduced. If these conditions exist then one would expect blood to flow backward more freely than in the normal heart. I have observed pinkening of vein and myocardium in the human heart with coronary disease after the graft has been placed and this pinkening has been marked.

So far I have performed this operation on sixty-two patients. The record is not entirely satisfactory. The one good feature about the record is that the operation is beneficial to patients with coronary artery disease. It is difficult to evaluate the status of coronary artery disease before and after operation. It is also difficult to make measurements of benefit on patients. How do we know whether the benefit that follows operation is due to the operation or would have occurred without the operation? This problem exists. I think I have seen enough good results to say that the operation is beneficial to patients with coronary artery disease. The mortality following operation depends upon the severity of the disease. The first twelve patients operated upon were totally or almost totally incapacitated. Eight of these patients died. Then we took a better type of patient and there was one death among 19 patients. Excluding the first twelve cases the mortality is 26 per cent. I think we could reduce mortality to about 10 per cent if we selected patients more carefully. However, it is the disease rather than the operation that contributes to mortality. Some of our patients died before the operation was done. In some of the patients the graft became thrombosed. In some the aorta was severely diseased so that the graft was not placed. I think we might be able to overcome the problem of thrombosis by cutting out a small piece of aorta as Dr. Bailey suggested. We used to do this in our early work but gave it up because it was not necessary on dogs and often the opening into the aorta became too large.

In conclusion, I should like to suggest that some of you surgeons become interested in this development. After some preliminary training on dogs the operation can be done satisfactorily on patients. The experimental work shows that the operation is beneficial. However, the final answer will come from the patients and will be decided upon through results as follows: (1) relief of pain, (2) ability to work, and (3) duration of life after operation. So far no patient who left the hospital with a patent graft has died.

DR. ARTHUR VINEBERG, Montreal.—I would like to take this opportunity to congratulate Dr. Bailey on his excellent presentation. I feel that the Beck method of revascularization of the heart seems to have possibilities and may even become the procedure of choice. It has, however, certain disadvantages and, for that reason, I should like to draw to your attention another method of revascularization of the myocardium, which I have presented before this Association on two separate occasions.

The method I refer to is internal mammary artery implant. You will recall that the left internal mammary artery is detached from the chest wall and placed within a tunnel in the

*This work is supported by a grant from the United States Public Health Service.

ventricular myocardium. We have shown in the past that this artery forms new branches which join with the left coronary circulation.

(Slide.) This shows an implanted internal mammary artery injected a few months after its implantation into the left ventricular myocardium. The injection mass was a rubber compound resisting acid digestion. The injection mass entered the internal mammary artery, was seen to spread through the anterior descending arteries of the left coronary, and come out of the anterior descending artery. A piece of polyethylene tubing was tied around the origin of the anterior descending branch left coronary and the whole specimen digested. The cast shown in this slide shows quite clearly an intimate junction between new branches of the internal mammary artery and branches of the anterior descending branch of the left coronary artery.

In all our injection specimens, the injection mass has entered the internal mammary artery and come out of a cannula placed in the left coronary artery, leaving little doubt that there is a true mammary coronary anastomosis.

(Slide.) It has been suggested by some that the anastomosis is superficial; this slide and the subsequent slide show the injection mass distributed through the entire left ventricular myocardium and from the inner to the outer surfaces of the heart.

It has also been suggested by some workers that the branches that are formed by the internal mammary artery are not truly arterial in character, but are capillaries which tend to disappear in a few weeks. This slide shows that the anastomosis is present up to fifty-eight weeks.

(Slide.) This slide shows an internal mammary artery which has been in the heart of a dog for over ninety days and represents a study made by the serial section of the anastomosed vessels with special stains for elastic tissue and muscle; note the elastic and muscle elements in one of the newly formed branches of the implanted internal mammary arteries. Serial section of seven different anastomoses has been thus studied with similar results.

Anatomic proof of a mammary coronary anastomosis is insufficient; for this reason functional experiments have been conducted to ascertain the value of the anastomosis. The first of these was that of survival studies following that of ligation of the left coronary artery.

(Slide.) A comparison is made between some of the figures after arterialization of the coronary sinus (Beck) and after internal mammary artery implant. It will be noted that from Beck's figures in the control series, there was a 70 per cent mortality rate with a 30 per cent survival with infarction. Whereas, after arterialization of the coronary sinus, there was a 10 per cent mortality and a 45 per cent survival with infarction and only 45 per cent survival without infarction.

In our own series, our control figures are approximately the same as those of Beck, namely, 80 per cent mortality and 20 per cent survival with infarction after internal descending branch ligation. Whereas in twenty-nine animals where there was a good mammary-coronary anastomosis, there were no deaths and all animals survived without infarction.

Others have published a much lower mortality rate for anterior descending branch ligation. The discrepancy between these figures and those obtained by Beck and ourselves I believe can be explained. The mortality rate is dependent on the number of branches tied, therefore it is necessary to be certain that the ligature placed around the anterior descending branch of the left coronary artery is located at its origin.

At autopsy, a careful search must be made for other branches arising from the left coronary artery itself which have not been included in the ligature and which could maintain life. A second experiment indicating the value of mammary-coronary anastomosis is shown on the following slides:

(Slide.) This animal survived an anterior descending branch ligation and six weeks later the internal mammary artery was tied off; the dog died forty-eight hours later with a large anterior wall infarct, indicating pretty clearly that the blood supply to this ventricle was largely supplied by the implanted internal mammary artery.

I am simply recalling this procedure to your attention. We have now operated on seven patients; there have been two deaths; three of the surviving patients are free of pain and are working; a fourth has been done recently. There has been one failure to date.

DR. HARRISON BLACK, Boston.—There are a few comments I would like to make on this paper. One point that should be borne in mind in the evaluation of any procedure for coronary insufficiency is that pain, although the most dramatic, is yet a poor criterion by which to judge the success of the operation. Many different events in our experience have resulted in the relief of angina without detectable prolongation of life. Among these are a dorsal sympathectomy, scarification of the epicardium, a myocardial infarction, or a period of shock. For this reason I believe we will have a better answer if we judge the various methods on the basis of survival. This admittedly is going to take a considerable period of time.

So far as experimental work is concerned, it seems to me that the crux of the problem is whether the procedure produces an increased blood flow in the capillary bed and thereby better oxygenation of the myocardium. This remains to be demonstrated conclusively for any of the current techniques and the method of Goldman mentioned earlier in this meeting may find application here. It is also worth noting that Palmer and Welch* have demonstrated that reversal of circulation in the lower extremity by femoral artery to femoral vein anastomosis is followed by deficient peripheral circulation, and they conclude that the end result differs little from a conventional arteriovenous fistula. The reason for this is that the blood is shunted through venous collaterals back to the heart, by-passing the capillary bed. Whether a similar situation obtains in the reversal of circulation procedures which have been described today remains to be seen. However, I think it is a possibility that must be seriously considered.

Another point to remember in evaluating experimental results is that in the dog survival rates following anterior descending coronary ligation can be improved by a variety of operative maneuvers in addition to the ones mentioned today. Furthermore, Schlesinger has been able to demonstrate by his injection techniques in human beings that a hemoglobin of less than 70 per cent for more than two months will produce intercoronary communications in more than 70 per cent of persons. It therefore must be proved that any procedure in question accomplishes more than these nonspecific stimuli.

I would like to mention an additional technique which we have used in the experimental laboratory and on two clinical cases. We have not felt that the approach introduced by Dr. Beck of producing vascular adhesions to the heart has been entirely exhausted. One of the observations we have made—and this is borne out by the work of Burchell at the Mayo Clinic—is that the epicardium is the barrier to the ingrowth of these vessels. We have found in the laboratory that if the epicardium is removed, however, vascularization of the myocardium is much improved. We have tried many methods of removing the epicardium using sharp dissection, bone dust, asbestos, trichloracetic acid, talc, aluronate paste, iodine, chlorine, etc. Only when 95 per cent phenol was used were we able to de-epicardialize the ventricle effectively without traumatizing the heart and thus obtain ingrowth of vessels into the myocardium which are large enough to be penetrated by the Schlesinger mass (lead phosphate in agar).

(Slide.) This slide shows a heart which has been painted with 95 per cent phenol. You can see the marked degeneration of the epicardium while the myocardium remains unaffected.

(Slide.) On the right are vascular adhesions which have been established following removal of the epicardium. On the left, new vessels have appeared in the myocardium and these are large enough to take the Schlesinger mass.

(Slide.) This is also a dog experiment, showing injection of the agar mass via the aorta as in the previous slide, with the proximal aorta clamped off so that the mass cannot

*Surg., Gynec. & Obst. 94: 206, 1952.

enter through the proximal coronary circulation. Above we see the bronchial arterial branches injected in the lungs, and in the heart itself there are vascular channels which are filled with the mass.

DR. BAILEY (Closing).—We have been very much interested in the work of Dr. Vineberg, have attempted to repeat his experiments in the laboratory, and have found some interesting corroborative evidence. However, we have not done enough yet to come out unequivocally and say that we have completely repeated them.

There is no doubt, as Vineberg has shown, that it is possible to place a large bleeding vessel into the myocardium and not get a hematoma. There is no other soft tissue that I know of that can offer that advantage to the surgeon. I think that observation in a sense begins to answer Dr. Black's comment about other organs in which reversal of circulation has not worked. The heart is of a peculiar spongelike structure, with big myocardial sinusoids and a rich capillary bed. It can drain off large quantities of blood and this drainage can compete with actively bleeding vessels.

It is true, as Dr. Black has pointed out, that pain is a poor criterion for the measurement of improvement in cases with coronary artery disease. So is dyspnea, because that also is a subjective symptom. Yet I note that all the eminent cardiologists who treat coronary artery disease do so on the basis only of subjective symptoms, except for the electrocardiogram. The ballistocardiogram may change that situation in the future. But at present all work tolerance tests are sharply limited by the onset of pain and dyspnea; so again they are actually subjective tests. I question them because we have been unable to evaluate these cases that way. Unless we hope to be better than the cardiologists themselves, we will have to accept subjective improvement as a therapeutic guide.

Those patients who had grafts which became thrombosed might be considered, in a sense, as controls to measure the effect of pericardial poudrage. There should have been improvement of pain. We put asbestos in the pericardium and certainly produced lots of pericardial adhesions. One of the patients does not have pain. All the rest do. They do not have the great improvement subjectively that the patients with open grafts present.

As to the test as to whether the myocardium actually does get better oxygenation, that is going to be quite a task and we have not figured out how that is going to be done.

The epicardium is, as Dr. Black has pointed out, a barrier to the formation of capillaries from pericardial adhesions. As you all know, Dr. Beck used to use a burr to take off the epicardium for that reason in his myopexy operations. He also used 90 per cent phenol, or perhaps it was 100 per cent, and twenty-seven other substances in the attempts to produce vascularized pericardial adhesions, in an experiment that was reported many years ago. Of them all, he felt that asbestos was by far the best available substance because it not only produced vascularized adhesions but also stimulated the development of the intercoronary communications.

I might say something about the electrocardiogram after arterialization of the sinus. The electrocardiogram in dogs and in human beings shows changes which might be interpreted as indicating a worsening of the condition. Since the dogs do not have coronary disease we do not think these changes have their usual significance. Therefore we believe that the one objective method cardiologists use for measurement of improvement in this disease is worthless for our purposes, because the electrocardiogram is apparently so greatly changed when the blood flow is reversed through the coronary sinus and its tributaries.

PULMONARY VALVULOTOMY

Description of a New Operative Approach With Comments About Diagnostic Characteristics of Pulmonic Valvular Stenosis

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SINCE the first successful pulmonary valvulotomies were performed by Brock[1] and Sellors[2] in 1948, this procedure has found increasing applicability in the treatment of congenital heart disease associated with stenosis of the pulmonic valve. Thus far, however, the total reported experiences with this method of treatment are relatively few. It is the purpose of this communication to describe a new operative approach, to make some comments about the diagnostic characteristics of pulmonic stenosis not associated with ventricular septal defect, and to present the results of treatment in a small series of cases.

THE OPERATIVE APPROACH

The operative procedure described by Brock[1, 3, 4] has proved generally satisfactory, and others[5-7] have followed it in essential detail. Use of the left anterior intercostal approach, however, has one potential disadvantage—the difficulty in certain cases of dislocating the heart into the left thorax so that traction sutures can be placed in the anterior wall of the right ventricle preliminary to cardiotomy and valvulotomy. This is particularly true in the less severe cases without marked right ventricular enlargement. Such difficulty was encountered in four of the first six valvulotomies performed through a left anterior thoracotomy. Indeed, in two of them, efforts at manual displacement of the heart into the left thorax were so regularly attended by alarming changes in cardiac rate and rhythm that it proved necessary first to place a suture in the wall of the presenting left ventricle and, by traction upon it, to expose the anterior right ventricular wall. As a consequence of these experiences the use of Milton's midline sternal-splitting approach was investigated. It was found to give excellent exposure and has been used with great satisfaction in the last three patients in whom a clear-cut preoperative diagnosis of pulmonic stenosis could be made. It has also been used in two additional patients who proved to have truncus arteriosus.*

The operation is performed in the following manner: The chest is prepared widely in the event the surgeon might wish to extend the incision into either

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*In one, pulmonic stenosis was considered a possibility because of known right ventricular hypertension without evidence of left to right shunt and inability to pass a catheter into the pulmonary artery. The other was thought to have tetralogy of Fallot. A side-to-end aortic pulmonary artery shunt using the left subclavian artery as a free graft had brought about considerable but incomplete improvement. An effort to perform a valvulotomy or infundibulectomy was considered safer than an attempt to produce an additional systemic-pulmonary artery shunt in view of the fact that one pulmonary artery had been divided and one subclavian sacrificed.

side of the thorax. An incision is made in the midline from the cephalad end of the manubrium to the lower end of the xiphoid. The incision is extended down through the periosteum which is stripped back from the midline a few millimeters on either side. The superior and posterior aspects of the upper end of the manubrium are freed sufficiently to permit engagement of the Lebsche knife. The sternum is split through its entire length. Any bleeding of the cut edges is controlled with bone wax or Oxycel or Gelfoam. The two halves of the sternum are retracted widely with one or two Rienhoff rib spreaders. A few cubic centimeters of 4 per cent procaine solution are injected into the pericardial sac. The lower portion of the thymus is dissected from the pericardium and reflected upward. The pericardium is incised in the midline after placing traction sutures in it, and the cut edges are retracted. Excellent exposure is obtained (Fig. 1). The pulmonary artery and the adjacent portion of the right ventricular wall are more or less in the middle of the field.

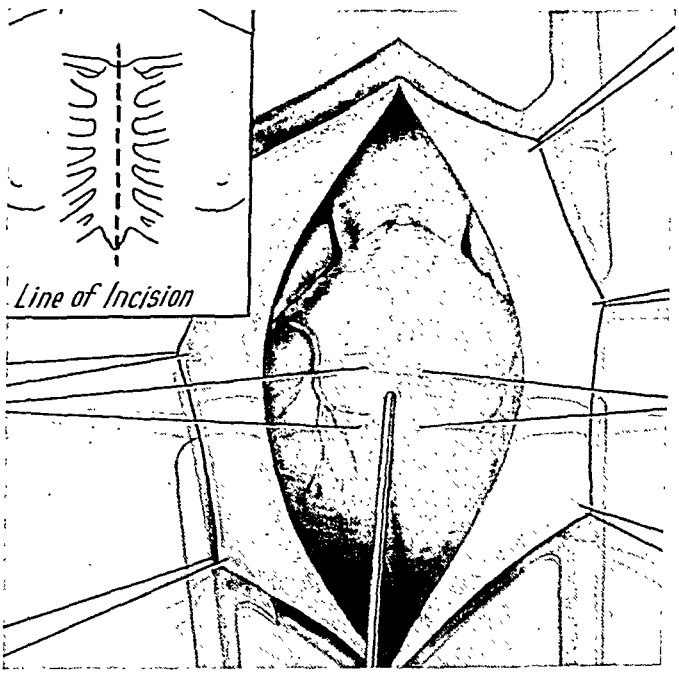


Fig. 1.—Drawing of the operative exposure.

The operation then proceeds according to the usual method. Procaine is injected into the ventricular wall at the point selected for cardiotomy. Traction sutures of silk are placed and a small stab wound is made. Exploration of the ventricle and pulmonary artery is carried out by passage of a small Hegar dilator. Either the Brock or the Potts valvulotomes are used for cutting the stenotic valve. Dilatation is carried out with the Potts dilator or by spreading a Kelly clamp. Bleeding from the ventricle is controlled by digital compression and by crossing of the traction sutures.

After completion of the dilatation the cardiotomy incision is repaired with a few interrupted silk sutures. The pericardial cavity is irrigated with normal saline solution and the lower portion of the pericardial incision is closed loosely with a few sutures of silk. That portion overlying the pulmonary artery is left open for drainage. Small Penrose drains are brought out through the upper and lower ends of the wound. If the pleural cavity has been inadvertently entered, the opening is enlarged and a catheter is introduced through an interspace. The sternum is approximated with a few sutures of stainless steel wire. In small children the sternum is soft and the sutures may be placed with a heavy cutting-edge needle. In older individuals they are passed through small drill holes. The periosteum, the deep and superficial fascia, and the skin are closed carefully with interrupted silk sutures. Upon return to the ward the patient is placed in an oxygen tent or is given oxygen through a nasal catheter. He is encouraged to lie in the prone position for the first few days in order to provide dependent drainage of the pericardium.

It is apparent that this approach is best utilized in cases in which preoperative studies point clearly to the diagnosis of pulmonic stenosis. Should the need arise it would seem feasible to extend the incision into either pleural cavity by transecting the appropriate half of the sternum and making an intercostal incision. Obviously in cases in which transthoracic exploration is felt to be of primary importance the sternal-splitting incision should not be used.

PULMONIC STENOSIS WITH INTACT VENTRICULAR SEPTUM

Pulmonary valvulotomy was performed in eight patients thought to have pulmonary valvular stenosis without ventricular septal defect. One patient was severely incapacitated. The others had mild to moderate disability (Table I). All had some dyspnea on exertion but only in two was it severe. All had fatigability, but it was extreme in only one. One patient had marked and five had only mild cyanosis. Cyanosis was not clinically evident in one patient and was only questionably present in another. Clubbing was extreme in one patient and mild in three; no definite clubbing was observed in four. All had a systolic murmur in the pulmonic area. A thrill was readily palpable in seven and suggestively present in the eighth case. In none was there a pulsating liver. The blood pressure tended to be normal or low. In Cases 2, 4, and 8 the systolic pressure ranged between 90 and 94. Hemoconcentration was very marked in one patient. Two had evidence of slight hemoconcentration, and there was no hemoconcentration in the remainder.

There was electrocardiographic evidence suggesting right ventricular enlargement in all except one case, but there was clear-cut evidence of right ventricular enlargement by fluoroscopic and radiologic study in only three and questionable evidence in two others. In three patients there was nothing to suggest specific chamber enlargement. In all except one there was enlargement of some portion of the pulmonary artery. In most of them the pulmonary trunk appeared dilated (Fig. 2). In several, however, there was no convexity in the cardiac segment representing the region of the pulmonary trunk but rather dilatation of the left or both main pulmonary arteries (Fig. 3). It was

TABLE I. SYMPTOMS AND SIGNS IN PATIENTS WITH PULMONIC VALVULAR STENOSIS AND INTACT VENTRICULAR SYSTEM

CASE NO.	AGE	SEX	DYSPNEA*	FATIGABILITY*	CYANOSIS*	CLUBBING*	R.B.C.	HEMATOCRIT	EVIDENCE OF RT. VENTRICULAR HYPERTROPHY		X-RAY EVIDENCE OF ENLARGED PULMONARY ARTERY	HIGH P WAVES	PERIOD OF FOLLOW-UP IN MONTHS
									X-RAY	ECG			
1	17	M	++	++	+	+	5.96	55	0	+	+	0	13
2	9	M	++	++	+	0	4.48	39	±	+	+	0	10
3	11	M	+	++	+	0	5.96	—	0	+	+	0	8
4	10	M	++++	++++	++++	++++	9.90	83	+	+	0	+	8
5	11	F	++	++	+	+	4.03	40	+	+	+	+	7
6	10	M	++	++	±	0	—	42	0	0	+	0	6
7	25	F	++	++	+	0	4.39	—	+	+	+	0	6
8	6	F	+++	++	0	±	4.10	—	±	+	+	0	2

*Listed according to severity from + to ++++; + indicating very mild, ++++ very severe.

felt by some observers that these pulmonary arteries were perhaps more translucent than would be expected in normal vessels of the same size. No intrinsic pulsation was noted in them fluoroscopically. One patient had a markedly enlarged heart. The others were either normal in over-all size or only slightly enlarged. The P waves were high in the electrocardiograms of only two of the eight patients.

Arterial oxygen saturation was determined in all (Table II) and cardiac catheterization* (Table III) in all save one. The exceptional patient was a diminutive boy of 10 (Case 4) who was extremely ill. He had marked cyanosis,

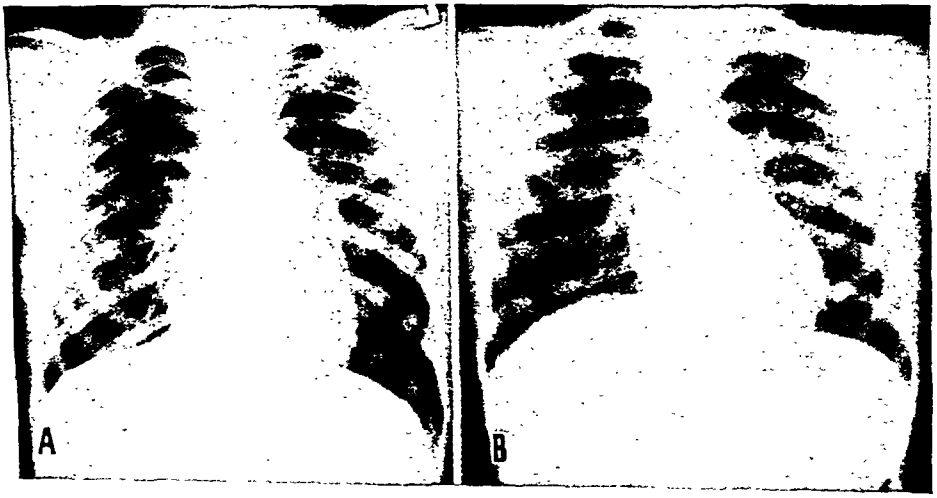


Fig. 2.—Roentgenograms of Cases 2 (*A*) and 8 (*B*) showing dilatation of the pulmonary trunk.

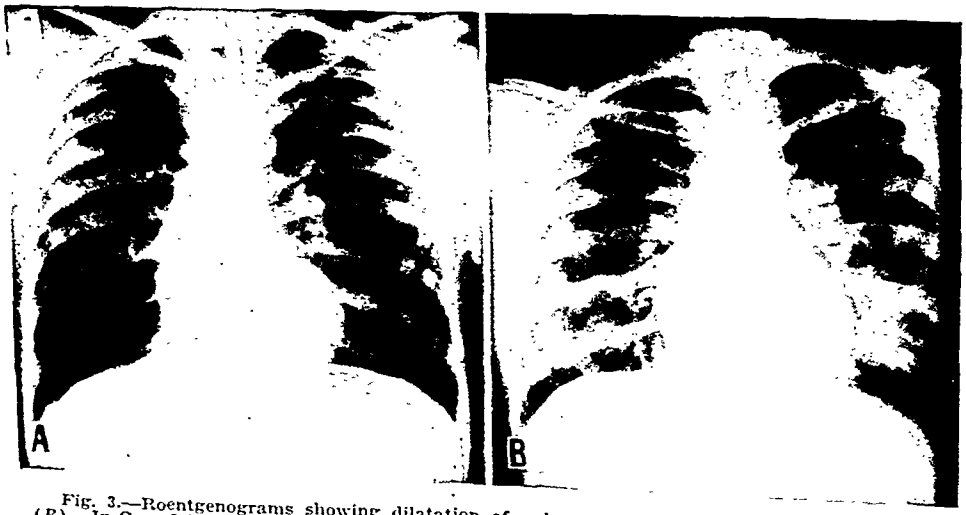


Fig. 3.—Roentgenograms showing dilatation of pulmonary arteries in Cases 3 (*A*) and 5 (*B*). In Case 3 it is clear that the region of the main pulmonary trunk is not enlarged. The left pulmonary artery is dilated, however. In Case 5 the main pulmonary trunk segment seems diminished in size but there is obvious dilatation of its main branches.

*The technique used is described in detail elsewhere.[3]

TABLE II. ARTERIAL OXYGEN SATURATION STUDIES IN CASES OF PULMONIC STENOSIS WITHOUT EVIDENCE OF VENTRICULAR SEPTAL DEFECT

CASE NO.	TIME OF TEST	BREATHING ROOM AIR	BREATHING OXYGEN LOW	BREATHING OXYGEN MEAN	BREATHING OXYGEN HIGH	CLASSIFICATION
1	Preoperative	89.0	89.2	90.9	92.6	Definite veno-arterial shunt
2	Preoperative	91.2	95.9	98.1	100.3	Borderline, possible small veno-arterial shunt
	Postoperative (8.5 mo.)	92.2	97.2	99.3	101.4	Borderline, possible small veno-arterial shunt
3	Preoperative	91.4	94.4	96.5	98.6	Definite veno-arterial shunt
4	Preoperative	56.9	61.9	63.2	64.5	Definite veno-arterial shunt
	Postoperative (3.5 mo.)	90.2	88.2	89.0	90.5	Definite veno-arterial shunt
5	Preoperative (1)	91.3	94.6	96.6	98.7	Definite veno-arterial shunt
	Preoperative (2)					
	Resting	89.3				Definite shunt
	Standing 5 min.	94.5				No shunt
	Exercise 1 min.	88.9				Definite shunt
	After exercise	96.8				No shunt
6	Preoperative	87.7	86.3	88.5	90.6	Definite veno-arterial shunt
	Postoperative (6.5 mo.)					
	Resting	94.3	98.1	100.5	102.9	Borderline, possible small veno-arterial shunt
	Exercise	92.6				
7	Preoperative	90.7	98.7	100.9	103.2	Definite lung factor, probably no veno-arterial shunt
	Postoperative (4 mo.)					
	Resting	91.7	100.8	103.1	105.5	Borderline, probable lung factor
	Exercise					Normal, probably due to exercise hyperventilation
8	Preoperative	91.1	94.3	96.7	99.1	Definite veno-arterial shunt

TABLE III. PRESSURE STUDIES IN CASES OF PULMONIC STENOSIS WITHOUT VENTRICULAR SEPTAL DEFECT

CASE NO.	PREOPERATIVE R.A.	PREOPERATIVE L.A.	PREOPERATIVE R.V.	PREOPERATIVE P.A., AT VALVE	PREOPERATIVE P.A., DISTAL	POSTOPERATIVE R.A.	POSTOPERATIVE R.V.	POSTOPERATIVE P.A., AT VALVE	POSTOPERATIVE P.A., DISTAL	PERCENTAGE FALL IN R.V. SYSTOLIC PRESSURE AFTER OPERATION
1	9 / 1	5 / −1	120 / 0	2 / −2	2 / −2					
2	—	—	65 / −7	10 / −1	16 / 4	—	39 / −5	12 / 2	18 / 6	40
3	16 / 2	21 / −3	82 / −6	24 / 6	46 / 5					
5	0 / −6	—	105 / 9	25 / 16	46 / 13					
6*	3 / −2	—	57 / 0	11 / 1	19 / 3	9 / 5	48 / 6	20 / 8	20 / 9	16
7	1 / −4	—	127 / −7	1 / 0	5 / 8	4 / −6	84 / −3	14 / 4		34
8	2 / −5	—	142 / 2	17 / 2	17 / 5					
By ventricular puncture										
Before valvulotomy			90 / 0							
After valvulotomy			64 / 0							

*Patient not in basal condition during postoperative study.

clubbing, hemoconcentration, and a large heart (Fig. 4). The liver was enlarged, heart sounds were faint, and he had a gallop rhythm. In this patient the resting arterial oxygen saturation was only 56.9 per cent. The remainder had only mild or questionable evidence of arterial oxygen unsaturation, the values ranging from 87.7 to 91.9 per cent. From these observations and values obtained during the breathing of pure oxygen, a definite right to left veno-arterial shunt can be presumed to be present in six and a possible small shunt in one additional patient (Case 2). In Case 7 it is clearly established that the arterial hypoxia was due to a lung factor and there was no definite evidence of veno-arterial shunt. None of the usual explanations for increased lung factor in congenital cardiac conditions were applicable in this case. In two of those with definite right to left shunt (Cases 1 and 3) the cardiac catheter passed readily into the left atrium and the right atrial pressure was elevated; it is

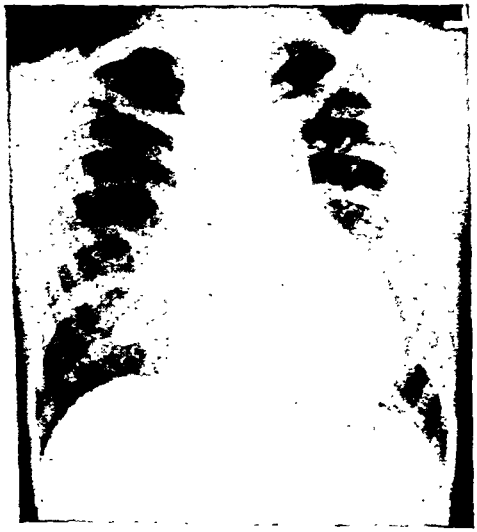


Fig. 4.—Roentgenogram in Case 4. The heart is seen to be greatly increased in size. There is evidence of right ventricular enlargement. The pulmonary artery segment does not appear remarkable.

entirely likely that a true atrial septal defect rather than a patent foramen ovale was present. That intermittent arterial hypoxia resulting from the intermittent shunting of blood from right to left atrium may occur in some is illustrated by studies carried out in Case 5. On one occasion the resting arterial oxygen saturation in this patient was 89.3 per cent. After standing for five minutes it was 94.5 per cent. At the end of one minute's exercise it was 88.9, and afterward 96.8 per cent. In none of the patients subjected to cardiac catheterization was there any evidence of a ventricular septal defect with left to right shunt from study of oxygen content of right atrial and right ventricular blood.

Pressure studies* made at the time of cardiac catheterization, and in one case by direct ventricular puncture at the time of operation, were of considerable interest. The right ventricular pressures ranged between 57 and 142 mm. of

*Made with the Hathaway manometer.

mercury. In one instance, at the upper extreme (Case 8), the systemic systolic pressure was exceeded by 40 mm. Right ventricular pressure was found to be near the systemic level in three patients (Cases 1, 5, and 7). In the other three patients the ventricular pressure was lower and was in the range in which as yet little experience with pulmonary valvulotomy has been obtained.

All of the pressures measured just beyond the pulmonary valve were lower than those measured more distally. This fact may be due to locally increased rate of flow with conversion of potential to kinetic energy at this point, followed by reconversion to potential energy more distally, in accordance with Bernouilli's principle. In five of the patients the pressure of the pulmonary artery near the valve was subnormal. In the two in which it was normal just beyond the valve, it was actually elevated more distally (Cases 3 and 5). The right atrial pressures were elevated in only two cases (Cases 1 and 3), in both of which entry into the left atrium was possible. The pressures in the right exceeded those in the left atrium during at least part of the cardiac cycle in these two cases. There was no frank right ventricular failure in any of the patients as evidenced by the normality of the ventricular diastolic pressures.

The single study by needle puncture of ventricular pressure at the time of operation revealed a fall in systolic pressure of about 30 per cent after valvulotomy. It will be interesting to learn whether the ultimate fall in ventricular pressure is predictable from such tracings made at the time of operation with the pericardium open. During preoperative catheterization the systolic ventricular pressure in this case was considerably higher than at the time of operation, 142 as compared with 90 mm. of mercury.

The diagnosis of congenital heart disease had been made early in life, usually before the age of 4 years. Most of the patients had apparently been thought to have an atrial or ventricular septal defect. The one patient in whom a diastolic as well as a systolic murmur was present had been considered as having a patent ductus arteriosus. Indeed, exploration had been carried out some months before the correct diagnosis was established.

During operation the pulmonary artery was found to be moderately or markedly dilated in all except one patient. In these cases the stenotic valve could be palpated through the pulmonary artery and a thrill could be felt with each systole. In the exceptional patient (Case 4) the pulmonary artery was small, having a diameter of less than a centimeter, and no thrill could be felt. The valvular stenosis in this, as in all the other cases, could be confirmed by palpation with probe and valvulotome, and the pulmonary artery began to pulsate vigorously immediately after valvulotomy and expanded to almost twice its former diameter. In one patient (Case 8) the valve was palpably calcified.

All of the patients have done well since operation. They have increased exercise capacity and, in general, are essentially free of symptoms within the limits of activity thus far permitted. When cyanosis was present beforehand it has disappeared or is milder in degree and less frequent in occurrence. The change in status following valvulotomy can be illustrated by those with the mildest (Case 3) and the severest (Case 4) symptoms.

The former patient was thought by his parents to exhibit dyspnea only rarely and after undue activity. Only infrequently was cyanosis of the lips noted, generally during periods of excitement. Though he could walk slowly for fairly long distances he tired easily and was always found to be sitting down to rest shortly after engaging in any play. Now eight months after operation he seems entirely normal. He plays vigorously, rides a bicycle well, and does not seem to tire at all.

The latter patient was so limited in exercise tolerance he could walk only a few steps at a time. He was markedly cyanotic and was forced to lead the exceedingly restricted life of a cardiac cripple. Because of poor heart sounds, gallop rhythm, and large liver, he was considered on the verge of heart failure and was digitalized before operation. From the moment of valvulotomy his color was excellent. He was discharged from the hospital on digitalis and with strict limitation of activity. About one week later he was readmitted in frank failure with marked edema probably resulting from excessive activity and too high a salt intake. He responded promptly to bed rest, salt restriction, and mercurial diuretics. Digitalis was discontinued a little less than three months after operation. Three and one-half months after valvulotomy the arterial oxygen saturation had risen from 56.9 to 90.2. It did not fall with mild sustained exercise (20 climbs up and down steps during a period of 2 minutes). The erythrocyte count had fallen from 9.90 to 4.43 million, the hematocrit from 83 to 52. He had gained from fifty-one to fifty-eight pounds and had grown in height. He now walks vigorously and has noted no fatigue on walking several blocks. His activity is being rapidly expanded.

In three additional patients postoperative arterial oxygen studies have been made (Cases 2, 6, and 7). All had higher values during breathing of air and during breathing of oxygen. Preoperatively one (Case 2) had a borderline condition with possible small veno-arterial shunt. The postoperative findings, though improved, were still in the same borderline classification. The second patient (Case 6) had evidence of definite veno-arterial shunt before operation. After operation the studies indicated a borderline situation with possible small shunt. The third patient (Case 7) had arterial hypoxia due to a lung factor before operation. Postoperatively she showed definite improvement but still had to be classified at rest as borderline with probably lung factor. During exercise after operation, probably as the result of hyperventilation and consequent increase in alveolar oxygen tension, the oxygen saturation rose to a definitely normal value. On cardiac catheterization these same three patients showed a reduction in right ventricular pressure, though not to normal levels, and a corresponding increase in pulmonary artery pressure.

It is of interest that a good result was obtained in the two patients in whom preoperative studies had demonstrated normal pressure values just beyond the pulmonary values and actually elevated pressures more distally. The outcome in one (Case 3) has been described. The other (Case 5) was digitalized during the postoperative period because of tachycardia. She has now been without digitalis for seven weeks and it is certain that she is considerably improved over

her preoperative state. The demonstration of pulmonary hypertension in two patients with pulmonic valvular stensosis is a surprising and, at present, unexplained occurrence. Our experiences demonstrate that, at least in mild degrees, it does not constitute a clear contraindication to valvulotomy.

OTHER PATIENTS TREATED BY VALVULOTOMY OR DILATATION OF THE PULMONARY ARTERY

Two additional patients have been treated by pulmonary valvulotomy and another by dilatation of a calcific stenosis of the pulmonary artery. One was a 21-year-old man thought to have tetralogy of Fallot. He had been severely incapacitated. Ten months earlier a left thoracotomy had been performed in the hope of establishing an aortic-pulmonary or subclavian-pulmonary shunt. The mediastinal area and the hilum of the lung were filled with a mass of wormlike collateral channels. In spite of care, bleeding was so troublesome that, alone in our experience with tetralogy of Fallot, it was felt necessary to abandon the attempt to isolate the pulmonary artery. A month later a segment of azygos vein was interposed as a free graft between the proximal ends of the divided subclavian artery and the upper lobe branch of the pulmonary artery. Though he was somewhat improved symptomatically and had less severe hemoconcentration, he was still badly handicapped and a continuous murmur was never clearly audible. Ten months after the first operation another exploration was made through the left thorax. At this time the hematocrit was 63. It had been 77 before the previous operations. Again the pulmonary artery could not be safely isolated. The lateral incision was extended anteriorly and a calcific stenosis of the pulmonary valve was discovered. Valvulotomy was carried out with good result. During the eleven months since operation he has been relatively well and has been working regularly. His color is excellent and the erythrocyte count normal.

A second patient was a pitifully handicapped 14-year-old girl with marked cyanosis, clubbing, hemoconcentration, physical incapacitation, and lethargy. From careful study including angiocardiography she was thought to have atypical tetralogy of Fallot with marked overriding of the aorta and unusually small pulmonary arteries. A left lateral thoracotomy was made. Preliminary dissection of the hilar area convinced the operator that if a pulmonary artery were present, it was diminutive in size. The incision was extended anteriorly and the pericardium opened. The main pulmonary artery was about normal in size or a little smaller than normal. The valve was not palpable. About 2 cm. distal to its origin, at about the region of the bifurcation, the artery became very small and there was obviously a calcified stenotic lesion in this area. A cardiotomy was performed and the calcific ring thoroughly dilated by spreading a Kelly clamp. Following this procedure, good pulsation was evident in the small left main pulmonary artery and a thrill could then be felt. The patient has been remarkably improved. On her first return visit three weeks after operation her color was good. She was much more energetic than she had been and could go up and down a flight of steps without dyspnea. Her activity is being rapidly increased.

A third patient was an 8-year-old boy with atypical tetralogy of Fallot which functioned essentially as a case of pulmonic valvular stenosis without ventricular septal defect. He had moderate dyspnea, fatigability, cyanosis, and clubbing but no hemoconcentration. The fluoroscopic and radiologic examination revealed right ventricular enlargement and decreased pulmonary artery shadows (Fig. 5). Angiocardiograms performed with anteroposterior and left anterior oblique projections revealed fairly large pulmonary arteries, suggestive evidence of infundibular stenosis, and normal sequential filling of the right and left sides of the heart, though some observers thought there was faint opacification of the aorta in one film at the time of right ventricular opacification. Cardiac catheterization studies revealed no increase in oxygen content in the right ventricle over that of the right atrium. A bubble in the system caused a slight damping effect with artificial elevation of diastolic, and lowering of systolic, pressures. Under the circumstances the right ventricular pressure was 67/9 and pulmonary artery pressure 15/6. Arterial oxygen saturation was 88. The patient was

explored for pulmonary valvulotomy. There was no visible abnormality in the region of the infundibular chamber. A jetlike systolic thrill was palpable in the pulmonary artery. No clear-cut stenosis was, however, palpable nor could one be sure of any infundibular stenosis. The Brock valvulotomes were passed and the pulmonary artery and infundibular chamber thoroughly dilated. The patient was considerably improved. He noted no dyspnea postoperatively, and his capacity for exercise was greatly improved. About thirteen months after operation he became acutely ill with a cold followed shortly by marked respiratory difficulty. He died within a few days. Post-mortem examination revealed the cause of death to have been massive left atelectasis from aspiration of foreign material which plugged the smaller bronchi. The heart showed right ventricular enlargement, a small high ventricular septal defect about 0.5 cm. in diameter with an overriding aorta, a moderate stenosis of the short infundibular chamber and the origin of the pulmonary artery, a large atrial septal defect, and drainage of the right pulmonary veins into the right atrium. There were one large and two small pulmonary valve cusps without fusion. The large valve cusp had been bisected by the valvulotome as had been a small cusp opposite it. There were linear scars in the infundibulum probably resulting from the passage of the valvulotome and the dilator. In spite of the complex malformation, catheterization and angiocardiographic studies demonstrated that the anomaly functioned essentially as a pulmonary stenosis with intact ventricular septum. It is of interest that marked improvement followed valvulotomy and dilatation and persisted during the thirteen months he lived.

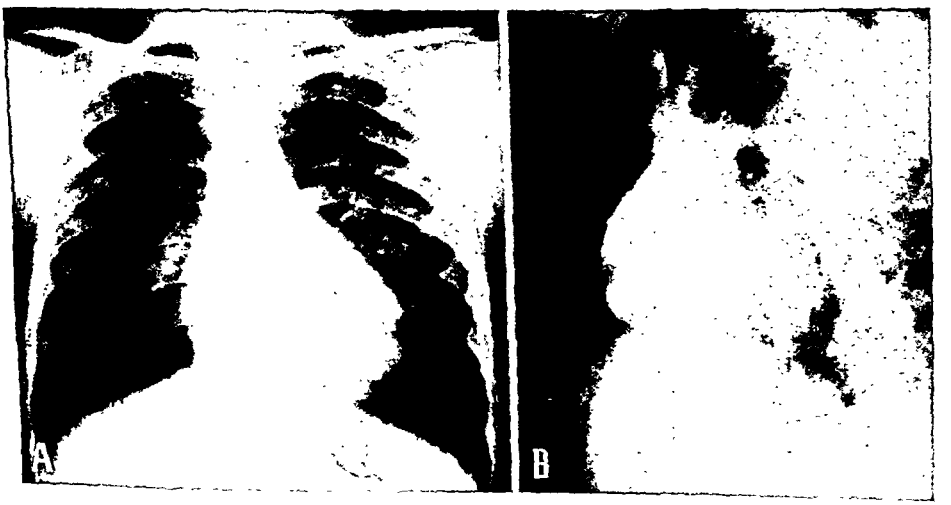


Fig. 5.—Roentgenograms in an unusual case of atypical tetralogy of Fallot. *A*, Plain roentgenogram showing right ventricular enlargement and diminished pulmonary vascular markings. *B*, Roentgenogram made shortly after intravenous injection of Diodrast. The pulmonary arteries are well visualized and there is no simultaneous opacification of the aorta.

SUMMARY

A new operative approach for pulmonary valvulotomy through a midline sternal splitting incision has been described. It gives excellent exposure of the anterior surface of the right ventricle and pulmonary artery and obviates the dislocation of the heart into the left thorax necessary when a left anterior thoracotomy is used in cases without marked right ventricular enlargement. A series of eight cases of pulmonic stenosis without evidence suggesting ventricular septal defect have been presented, and the diagnostic characteristics of this condition are presented with particular reference to the milder cases without the

classical signs noted in those with more advanced alterations. Good functional results were obtained even when the right ventricular pressure was not elevated to extreme levels and when mild pulmonary hypertension was present. The results of arterial oxygen and catheterization studies have been discussed. Three unusual cases of tetralogy of Fallot in which the patients were subjected to valvulotomy or pulmonary artery dilatation have also been presented.

Thus far there has been reported a total of sixty-two operatively treated patients with pulmonic valvular stenosis without ventricular septal defect, of which fifty-four survived and were benefited by the procedure.[2, 4-7, 9] Adding our eight cases in which no deaths occurred, seventy patients have been treated, with a mortality of 10 per cent. It is believed that the mortality will prove to be much lower in the milder cases of pulmonic stenosis and that it is worth while to continue exploration of the problem of their surgical treatment.

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DISCUSSION

DR. OSLER ABBOTT, Atlanta.—I am deeply appreciative of the physiologic understanding and thought involved in this paper. I want to bring up two aspects which I think are of some importance. One of the great features of a good surgeon, or of a surgical procedure, is adjustability, and I think it is important to stress that there is an optimum approach to every organ and structure. There is, however, a terrific adjustability in the thorax, and pulmonic stenosis can be approached from a lateral incision if the surgeon will adjust himself to moving from the left side of the table to the right, from an anterior incision and from a median sternotomy. Of course, much of the future of surgery is the proper combination of useful procedures, including the mixing of one incision with the other. I think that this is important. A tetralogy can be explored where it is planned to do, for instance in the young age group, a side-to-side anastomosis of the Potts type. The clinical judgment may not be found good, and a switch may be made to a valvulotomy, still using the lateral incision. With the lateral approach, if the surgeon will work facing the patient's anterior thorax as he lies in the left lateral decubitus, he will have good access.

The second point I would like to make is one on which I have had some correspondence with Dr. Blalock, and that is the question of anesthesia in pulmonary stenosis surgery. The question in my mind has been, if we give some positive pressure prior to opening the thorax, do we not increase pulmonary resistance, or alveolar tension, produce reflex pulmonary artery and bronchial artery spasm and therefore indirectly produce cardiac anoxia? In pulmonary

valve surgery the unfortunate frequency of cardiac standstill at the beginning of the operation is a phenomenon which so many of us have encountered, and Mr. Brock has so well emphasized. He insists that the surgical approach to these pulmonary stenosis patients must not be delayed and if difficulty develops, the valve must be opened quickly. The improvement of the patient is often striking as soon as the thorax is opened. Since that talk about three years ago, we have made a markedly strong point, that no positive pressure is given to a patient with obstruction of the right ventricular outflow tract until the thorax is opened. I would be interested to see what the results of such a concept would be if applied by a larger number of surgical clinics.

DR. LEO J. KENNEY, Detroit.—At the Henry Ford Hospital we have carried out a pulmonary valvulotomy in a total of ten cases, and there has not been any mortality in the series. We have a great interest in this subject.

I would like to present a simple diagnostic test devised by Dr. Robert Ziegler of the Division of Pediatric Cardiology, Henry Ford Hospital, which has given us considerable aid in the diagnosis of this entity and also in the diagnosis of the other congenital cardiac anomalies.

(Slide.) The test is carried out at the time of cardiac catheterization, after the physiologic data have been obtained. The tip of the catheter is permitted to remain in the right ventricle and 2 c.c. of 2 per cent sterile fluorescein dye is injected through the catheter into the right ventricular chamber. The period of time required for the dye to appear at the mouth is determined by a stop watch and an ultraviolet light directed at the mucous membrane of the patient's lips. A definite end point is obtained and identified by the characteristic appearance of fluorescein. Normally, four to five seconds are required for the dye to go from the right ventricle through the lungs, back into the left side of the heart, and out into the systemic circulation to the lips. In patients with pure valvular stenosis this time may be prolonged up to fourteen seconds. In the instance of a tetralogy of Fallot, with the patent interventricular septal defect, the dye appears in one to four seconds. This test is in routine use at our hospital and has been applied in over 200 cases without any harmful effects. Details have been published in a recent issue of the journal of *Circulation* by Dr. Ziegler.

DR. H. WILLIAM SCOTT, JR., Nashville.—I would like to congratulate Dr. Shumacker on these interesting studies and on his good results in these patients with valvular pulmonic stenosis. In the first direct attack on a stenotic lesion of a pulmonary valve, which was done at Johns Hopkins Hospital, Dr. Blalock used a sternum-splitting incision and the exposure was quite satisfactory. So far as I know, since that time exploration has been done in all subsequent cases through an anterior intercostal incision on the left. When the heart is large this exposure is quite satisfactory, but when it is small, as Dr. Shumacker has pointed out, there are some objections to this approach. It may be necessary, under these circumstances, to rotate the heart to the left by traction on the medial flap of the pericardium and that, certainly, is not entirely without ill effects. In such instances I have always had in mind the extension of the intercostal incision transversely across the sternum to gain adequate exposure of the base of the right ventricle and feel that with this additional maneuver, which may not be necessary except on rare occasions, the anterior intercostal approach is satisfactory for routine use, and in this use it may disturb the patient less than the use of a sternum-splitting procedure.

DR. HENRY SWAN, Denver.—I would like to put in an additional plug for the sternum-splitting maneuver. We have been splitting the sternum transversely in patients in whom it seemed desirable to have a wide exposure of the heart and access to all chambers. The incision has been in the fourth interspace on the right, entering the pleural cavity, across the sternum in a jog formation, and out the fifth interspace on the left. This little jog is put in to stabilize the postoperative repair of the sternum. We have encountered four patients with intracardiac foreign body, two with constrictive pericarditis, and one with an aneurysm of the ascending aorta through this incision, and it certainly provides a very wide and satis-

factory exposure. I would agree with Dr. Scott that in patients with pulmonic stenosis, if the left intercostal incision is placed, going across the sternum in cases in which it was found necessary, this would certainly give a very adequate exposure.

DR. RALPH A. DETERLING, JR., New York.—At Presbyterian Hospital in New York, in Dr. Humphreys's group, we have performed about twenty Brock operations with no mortality. We have employed the left anterior approach and have as yet had no great difficulty. I would agree with Dr. Swan and Dr. Scott in the desirability of being able to carry out a transsternal splitting if it did seem necessary. The main reason I wish to speak, though, is to mention the findings in two interesting cases we had in this group of pulmonic stenosis. At operation, direct intraventricular and arterial pressure are obtained by fine plastic catheters introduced through a small needle into the lumen and connected to a Lilly manometer. We have carried out perhaps 200 such studies in operations on the heart and great vessels, and have found several in which the findings at operation have been extremely important in influencing the surgical technique. In one of Dr. Humphreys's patients, the standard Brock procedure had been completed. A study of the pressures obtained with the catheter, about 1 mm. in diameter, demonstrated to our surprise that there was relatively little change in the pressure of the pulmonary artery, which was quite low, or in the elevated right ventricular pressure. So with some hesitation, as you can imagine, he entered the right ventricle and did a much more vigorous dilatation of the valve. The pressure tracings then indicated restoration toward normal of these pressures.

In a second patient upon whom I operated, we were studying the pressures at the end of the operation and were somewhat surprised to find the fall in the right ventricular pressure to be much less than was the rise in the pulmonary arterial pressure. Upon introducing the catheter into the right ventricle, I was informed by our associates that they had obtained an aortic pressure and pulse wave contour. The tip of the catheter had gone through an unsuspected high interventricular septal defect. Actually, the Brock procedure was allowing a more effective left to right shunt by opening the right ventricular outflow tract.

I think we should continue with such studies, since we are actually exploring the field of human cardiovascular physiology at the present time.

DR. SHUMACKER (Closing).—I certainly appreciate the comments of those who have discussed our paper. We have used the fluoroscein test which Dr. Kenny mentioned but have not found it entirely reliable. Certainly any procedure which would increase the accuracy of diagnosis would be of great value. One case, which is described in the manuscript but was omitted from the presentation for the sake of brevity, illustrates clearly the difficulty which one may encounter in diagnosis. This boy was eventually proved to have an atypical tetralogy of Fallot. According to cardiac catheterization studies and angiocardiography his malformed heart functioned in a manner quite like that of pulmonic stenosis and he was considerably improved after pulmonary valvulotomy and dilatation of the infundibulum and the pulmonary artery.

I am sure that the comments which have been made about the extensibility of various thoracic incisions are sound. We too have done explorations on several patients through lateral thoracotomies and, when we found it necessary to open the pericardium and perform pulmonary valvulotomy, have simply extended the incision anteriorly and completed the procedure satisfactorily. We are at present using the sternum-splitting approach in seemingly proved cases of pulmonic valvular stenosis because we find that it is so easy in execution, that it provides such excellent exposure, and is so easy to repair. It seems to me that it is an approach which others may profitably employ in cases of this sort. It is of great interest to me to learn that Dr. Blalock used it in his first case of pulmonary valvulotomy.

PLASMACYTOMA OF THE LUNG

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EXTRAMEDULLARY plasmacytoma is a relatively rare tumor and in the literature there exists much confusion concerning its origin and behavior. A survey of the literature shows that about 185 cases of extramedullary plasmacytoma have been reported since 1905. Hellwig[1] reviewed 127 cases in 1943 and added one of his own. This represented all cases reported from 1905 to 1943, with the exception of 24 cases reported by Jaeger[2] in 1942. Since that time a number of cases have been added and several reviews of the subject have been presented.

In all of these, only four extramedullary plasmacytomas were reported which were thought to have originated in the lung. The first was reported by Gordon and Walker in 1944.[3] There is little doubt that this is a plasmacytoma of the lung. The patient survived left upper lobectomy, but there is no further report. The second patient with a plasma cell tumor believed to be in the lung was reported by Kuley and Kuntman.[4] The lesion in their patient, however, is not conclusively in the lung. The roentgenograms presented with the report show that the lesion could well have originated from the mediastinum. The diagnosis rests only upon material obtained by aspiration; therefore, it is difficult to accept this as a plasmacytoma of the lung. The third report by Childress and Adie[5] describes a plasmacytoma in the right middle lobe of a 21-year-old white man. It was removed by segmental resection. At the time of their report the patient had been followed for three years and apparently was doing well. The fourth report by Cotton and Penido[6] describes a 57-year-old woman who survived left upper lobectomy for plasmacytoma and who was well some twenty months later at the time of the report.

Because of the obvious rarity of this condition, and because of other unique features, it was thought useful to report a plasmacytoma of the lung which came under our observation. We believe this to be the fourth confirmed plasmacytoma of the lung. It is the youngest patient (age 3 years) on record with extramedullary plasmacytoma in any location. It represents the longest follow-up (eight years) of any plasmacytoma of the lung.

CASE REPORT

History.—D. C. S., University of Virginia Hospital No. 202614. This 3-year-old white girl was admitted to the Pediatric Service at the University of Virginia Hospital on June 6, 1944, with generalized swelling and dyspnea of one year's duration. She had been well for the first two years of her life, and one year prior to entering the hospital she became fretful and lost her appetite. Later she developed generalized edema, and her urine became dark. These signs persisted until admission. During the year she had several bouts of fever accompanied by increase in the edema. She also had exertional dyspnea which became progressively worse. During the winter months, the patient had developed an intolerance

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to cold, and became cyanotic on the slightest exposure to cold. Six months prior to admission, the parents noted clubbing of the fingers which became more prominent until admission. There was no cough, hemoptysis, expectoration, or specific chest pain. Her past history revealed that she had been born by a normal delivery, had never been cyanotic as an infant, had an entirely normal development, and was very energetic until the onset of her illness.

Physical examination on admission revealed a temperature of 103.6° F. rectally, pulse rate of 120, respirations 40. She weighed 14.6 kg., and appeared to be a well-developed 3-year-old girl with 2 plus pitting edema of the arms, legs, and face. She was in no distress. There were shotty, nontender, discrete nodes in all of the regional areas. The spine showed a moderate dorsal kyphosis. There was a moderate increase in the anteroposterior diameter of the chest, and respirations were almost entirely abdominal. There was dullness to percussion over the left hemithorax from the third rib anteriorly to the third thoracic vertebra posteriorly, with decreased breath sounds over the same area. There were no râles. Examination of the heart revealed a diffuse forceful impulse, most intense below the sternum at the level of the fifth costal cartilage, which gave the impression that the heart was pushed to the right. There was a harsh systolic murmur over the entire precordium. The neck veins were distended, and there were many enlarged cutaneous veins over the thorax, arms, and legs. The liver edge was 2 to 3 fingerbreadths below the right costal margin. The spleen was readily palpable. There was slight abdominal distention. The knees were swollen, fluctuant, and painful on motion. There was marked clubbing of the fingers and toes.



Fig. 1.—Preoperative roentgenogram showing anteroposterior view of dense mass occupying nearly the entire left hemithorax.

Laboratory Data.—Hemoglobin—8 Gm. (Haden-Hauser); red blood cells—4.3 million; white blood cells—7,600, with a normal differential. The venous pressure in the right arm was 150 mm. of water. The blood Wassermann and Kahn were negative. Blood urea was 22 mg. per 100 c.c. Total protein was 5.9 Gm. per cent, and a single blood culture was negative at forty-eight hours. The urine was yellow, slightly cloudy, with 4 to 8 white blood cells and 10 to 15 red blood cells per high-power field, with no casts. It was negative for Bence-Jones protein.

Roentgenograms of the chest revealed a very large rounded opacity which nearly filled the left hemithorax. The mass occupied the posterior portion of the chest to within 1 to 1.5 cm. of the anterior thoracic wall (Fig. 1). There were no definite pulsations on fluoroscopy. The

heart and most of the mediastinum were quite markedly displaced to the right. Roentgenograms of the extremities revealed almost the total length of the shafts of all the long bones to present marked periosteal new bone formation which was sharply demarcated from the underlying cortex. The peripheral margins of the bones were also sharply delineated. The mass suggested the possibility of a large fluid-filled pulmonary cyst, a walled-off pocket of fluid, or possibly a huge intrathoracic neoplasm. The bone changes were typical of a marked degree of pulmonary osteoarthropathy.

Aspiration biopsy of the mass was performed. Examination of the material obtained revealed a solid tumor made up of large masses of spindle-shaped cells with hyperchromatic nuclei. Mitoses were thought to be moderately increased. Some palisading of the cells was noted. The exact type of tumor could not be determined from such a minute biopsy, but it was thought that the tumor might represent a sarcoma or lymphoblastoma.

It was thought from the histologic appearance that the tumor might respond to x-ray treatment. This treatment was delayed because the patient developed measles, which subsided in four days. Three days later roentgen therapy was begun over the left hemithorax; 2,800 r in air (filtration—2 mm. Cu) being given through two portals over a period of fourteen days. Following this a roentgenogram of the chest revealed the tumor to have increased in size and to have extended to the first interspace. Because of this failure to respond to therapy, it became obvious that thoracotomy offered the patient her only chance for cure. On July 10, 1944, exploratory thoracotomy was carried out by one of us (M. L. W.). The pleura was freed, and a large dense mass was found occupying all of the lower lobe on the left and extending across the interlobar fissure into the upper lobe. There appeared to be no normal lower-lobe tissue remaining. Exploration of the mediastinum revealed it to be entirely free of tumor. Pneumonectomy was carried out using the individual ligation technique. One lymph node was removed for examination, and grossly appeared benign. It was felt that the entire tumor mass had been removed. The patient tolerated the procedure nicely, and had an essentially uneventful postoperative course.

On gross examination, the tumor in the left lower lobe measured 7 by 7 by 8 cm. It was yellowish-brown and fairly soft. It seemed to have a definite capsule, and was quite circumscribed on cut section. It did not appear to arise from a bronchus. The hilar region was not involved. On microscopic examination, the tumor was composed of a stroma of spindle-shaped cells growing in loose strands. These cells had blunt, rounded nuclei, and were fairly uniform in structure and staining properties. The predominant cells, however, in most fields were round, oval, or polygonal cells with abundant cytoplasm and eccentric nuclei containing from five to ten distinct deeply-staining blocks of chromatin regularly arranged in a circle within the nuclear membrane. Adjacent to the nucleus there could be found the characteristic "paranuclear light-stained" area. This predominating cell fulfilled the criteria for the diagnosis of plasma cells as outlined by Marschalko.[7] A search was made for Russell bodies but none were found (Fig. 2).

The patient's postoperative course was essentially uneventful. She was discharged on Aug. 6, 1944, in good condition. She first returned for follow-up examination on Oct. 2, 1944. There was an opacity in the left hemithorax, with a fluid level and thickening of the pleura. The periosteal new bone formation and the obvious signs of advanced osteoarthropathy were noted to be subsiding. The patient was then asymptomatic, and gained weight for one year. A small mass in the right lung was noted in 1945 and was followed. By Jan. 31, 1946, it was noted to have decreased in size and become less distinct. It was thought to be a typical Ghon tubercle. The osteoarthropathy had completely disappeared by Jan. 1, 1946. Radiographic follow-up of the chest during and subsequent to 1946 show clearly that there were lung markings filling the entire chest and on examination the patient had good breath sounds over the operative side (Fig. 3). At last examination (March, 1952) the patient was entirely well. She was able to run and play with other children and had no detectable respiratory limitations. Her respiratory studies show a maximum breathing capacity of 30.8 L per minute, with a vital capacity of 1,346 c.c. The patient is gaining weight and doing well in school. Expansion of the operated side on respiration is good, and nearly equals that of the right side. The breath sounds on both sides are equal. Filling of the entire thorax by the remaining lung following pneumonectomy has been noted in other patients. It is unusual, however, to find so little distortion in the mediastinum.



Fig. 2.—Photomicrograph (hematoxylin-eosin ×1200) showing the predominating typical Marschalko plasmacytes.

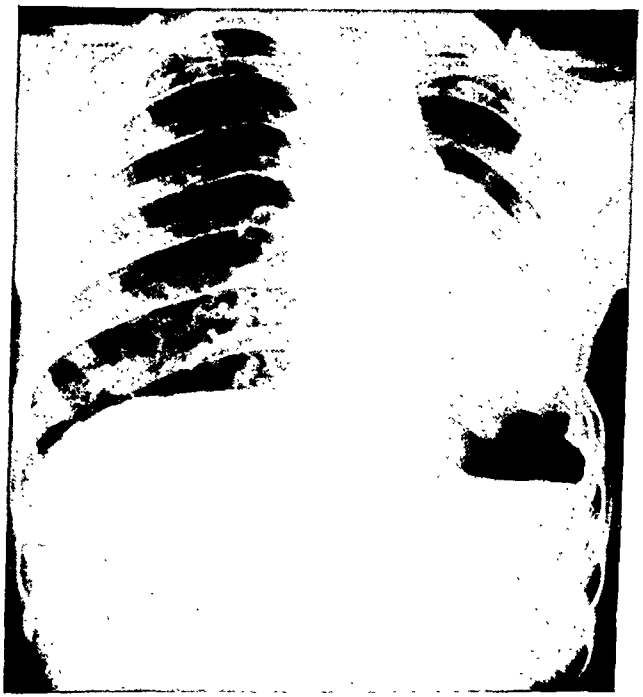


Fig. 3.—Roentgenogram showing anteroposterior view of thorax eight years after left pneumonectomy. Faint but definite lung markings can be seen in the left hemithorax.

DISCUSSION

Recent work dealing with the plasma cell by Jordan[8] has established some new information concerning the nature of the plasma cell, and has confirmed previous data dealing with the origin of this cell. Studies by Jordan of tissue explants, consisting almost solely of lymphocytes, have clearly shown that lymphocytes differentiate into plasmacytes. In many tissue explants containing lymphocytes, the predominating cells after five to six days were typical Marschalko plasmacytes. In addition, the plasmacytes were found capable of extensive ameboid motility and complete amitotic division. The latter was confirmed in cinematographic records. These actual cinematographic records confirm the tissue culture work done by Maximow[9] in 1922, which showed the derivation of plasma cells from lymphocytes. In spite of Maximow's work, there has been considerable argument concerning the origin of the plasma cell. Originally, Ramon y Cajal, who first described the plasma cell in 1890, contended that the plasma cell originated from fixed tissue lymphocytes. Unna contended that plasma cells are derived from fixed connective tissue elements. Marschalko believed the origin of plasma cells to be from emigrated blood lymphocytes only. Maximow and Jordan's works appear to present definite evidence that plasma cells may be derived from both fixed and emigrated lymphocytes. Since the plasmacytoma may originate from lymphocytes, it may bear some relation to tumors of lymphatic tissue.

It has been maintained by some that extramedullary plasmacytomas are granulomatous in nature, and consequently are benign. However, a review of the clinical experience with these tumors shows this to be a dangerous viewpoint. Hellwig found that out of 128 cases reported in his review, 29 showed definite invasion locally. Thirteen of these showed distant metastases. Figi, Broders, and Havens[10] reported eleven cases of plasmacytoma in 1945. Five of the eleven cases showed frankly malignant behavior with either extensive local invasion or distant metastases. On the basis of their experience they have stated: "Plasmacytomas are malignant lesions and they should be classified and treated as such." They found no evidence from their material to suggest that these lesions are granulomatous or inflammatory in nature. In 1946, Couret[11] reported a plasmacytoma of the pyloric portion of the stomach. This lesion was locally resected. It was found to be a plasmacytoma, and was thought to be an inflammatory benign lesion. Two months later the patient was explored for obstruction, and was found to have large fungating masses throughout the abdomen which proved to be composed predominantly of lymphocytes. One month later, autopsy revealed the large definitely invasive masses to represent plasmacytoma. Shaw and Smith[12] reported a plasmacytoma of the thyroid gland which was almost exclusively made up of plasma cells with a moderate number of mitoses suggesting a low-grade malignancy rather than an inflammatory lesion. Many other cases can be cited which show definitely that a high percentage of plasmacytomas become malignant locally and give rise to distant metastases. It should be stressed that the microscopic picture of these tumors cannot be correlated with their clinical behavior. These cells have been shown to be capable of amitotic division and in tissue cultures have been noted to multiply by this means in

most instances rather than by mitotic division. It is easy to see, therefore, why the absence of mitoses in these tumors has misled many observers into classifying the lesion as benign, only to have the lesion become frankly malignant. It is obvious that plasmacytomas cannot be classified on the basis of their microscopic appearances in the same manner as carcinomas.

Gross appearance may also be misleading. Apparent encapsulation and sharp delineation from surrounding tissue have been noted in lesions which have given rise to metastases. Because plasma cells have been noted to be abundant in some inflammatory diseases, particularly syphilis, tuberculosis, and neisserian infections, many observers have regarded practically all lesions containing plasma cells as granulomas. These inflammatory lesions should be sharply separated from plasmacytomas such as the one reported here, in which there is little or nothing to suggest an inflammatory lesion.

TREATMENT

Sixty-five per cent of the reported cases have been treated by surgery alone, about 18 per cent have been treated by a combination of surgery and radiation, and the remainder have been treated by radiation alone. Some of the tumors reported were apparently radiosensitive and good results were obtained but a number of others failed to respond to roentgenotherapy as did our case. Surgical treatment was then utilized when it was still feasible. In view of the good results obtained in our one patient and the good immediate result obtained by surgical means in the other three plasmacytomas of the lung, surgery would certainly appear to be the treatment of choice when the lesion is pulmonary in origin. According to the results obtained in treated cases reported in the literature, it would seem that surgery is also the treatment of choice in those tumors elsewhere in the body that are accessible to removal. A number of these tumors have recurred, however, despite surgery and radiation combined. Apparently the best results are obtained in tumors located in the conjunctiva. The conjunctival tumors are benign lesions with few exceptions, though Tajkef[13] reported a case of plasmacytoma of the conjunctiva which recurred repeatedly after several excisions. It has probably been these tumors of the conjunctiva that have led some workers into a misconception concerning plasmacytomas. In other locations in the body, the tumors have by no means been benign. The experiences of Couret, Shaw and Smith, Figi and Broders, and others testify to the highly malignant behavior of a large percentage of extramedullary plasmacytomas. Certainly, it is safe to say that our knowledge of these tumors is limited, and at present there is no means of determining in an individual case whether a plasmacytoma is benign or malignant provided, of course, metastases are not already obvious. Until we are able to make this determination in a given case, we are inclined to agree with Figi and Broders that these tumors should be regarded as malignant and treated accordingly.

SUMMARY

A case of plasmacytoma of the lung in a 3-year-old girl is presented. A left total pneumonectomy was done, and the patient has been followed for eight

years and has no evidence of recurrence or metastasis. This is apparently the fourth reported case of plasmacytoma of the lung proved clinically and pathologically. A follow-up of eight years is presented with complete regression of advanced pulmonary osteoarthropathy and complete filling of the thorax by the remaining right lung. A brief review of the literature is presented in which it is pointed out that these tumors in many instances are malignant. Surgical removal is the treatment of choice in most cases of extramedullary plasmacytomas outside the conjunctiva, provided there has been no metastasis or invasion of vital structures.

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PHYSIOLOGIC STUDIES FOLLOWING THORACIC SURGERY

II. Immediate Effects of Upper Lobectomy Combined With a Five-Rib Thoracoplasty

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INTRODUCTION

THE first paper in this series reported on the immediate physiologic effects following each of three stages of a seven-rib thoracoplasty.[1] The present work deals with a similar study made following a one-stage operation in which an upper lobe was resected and a five-rib thoracoplasty was performed.

In the search for a satisfactory procedure for the treatment of cavitary and nodular active tuberculosis, many surgical approaches are being tried by various groups. The combination of a five-rib thoracoplasty and an upper lobectomy was devised to allow extirpation of the principal site of the disease without overexpansion of the lung tissue remaining in the chest.[2-4] The thoracoplasty was performed immediately after the lobectomy and as a part of the same procedure. A subsequent report will cover the effects of segmental and wedge resections. Since resectional procedures are relatively new, it was felt advisable to obtain accurate observations upon patients subjected to such operations and to compare the effects of the various procedures with the effects of a thoracoplasty. Thoracoplasty was selected as a standard for comparison, since the clinical effects of this operation are so well known.

It is to be emphasized that this study was concerned only with the immediate physiologic effects of the various procedures. No consideration was given to the effects upon the tuberculous disease.

METHODS

The methods of study employed in this group of patients were the same as those reported in detail in the first paper of this series.

CLINICAL MATERIAL

Eleven male patients ranging in age from 20 to 59 years were selected for study. Each had previously been reviewed and accepted for surgery by members of the medical and thoracic surgical services. Each patient had predominantly unilateral, upper lobe, cavitary and nodular tuberculosis. The contralateral lung was either clear or contained apparently stable tuberculous lesions. In one case (N. H.) there was pleural thickening over the lower and middle lobes on the involved side as a result of a previous therapeutic pneumothorax.

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In this patient the middle and lower lobes were decorticated at the time of the lobectomy-thoracoplasty. In one subject (H. R.) the right middle lobe was also removed because of nodular disease. In this same case there was evidence of pleural thickening over the contralateral lung as a result of a previous therapeutic pneumothorax. This case will be discussed in detail below. One patient (L. D.) had silicosis (Stage II) throughout all five lobes, but functionally he did not constitute a special problem. Only one patient (W. H.) manifested any dyspnea on the exertion of ascending a flight of stairs. The comparison of the preoperative pulmonary functional studies with those of a group of 16 normal residents is given in Table I. The only significant abnormalities were a decrease in maximum breathing capacity, an increase in alveolar retention of nitrogen after seven minutes of breathing pure oxygen, and an increase in the residual volume (Table I).

TABLE I. THORACOPLASTY AND LOBECTOMY

	MEAN NORMAL	MEAN PREOPERATIVE	MEAN POSTOPERATIVE
Oral temperature (°F.)		98.2 ± 0.55	100.3 ± 1.25*
Pulse rate/min.		78.0 ± 12.5	116 ± 16.8*
Metabolic rate $\frac{\text{(cal./hr.)}}{M^2}$	35.0 ± 2.7	38.8 ± 5.0	47.0 ± 5.7*
Respiratory quotient	0.90 ± 0.043	0.80 ± 0.08	0.76 ± 0.05
Arterial blood:			
pH	7.40 ± 0.01	7.39 ± 0.01	7.37 ± 0.01
CO_2 content mm./L.	21.30 ± 1.0	22.37 ± 0.95	22.49 ± 2.4
P_{CO_2} (mm. Hg)	39.0 ± 2.0	43.3 ± 3.0	46.0 ± 6.1
Hemoglobin saturation (per cent)	96.5 ± 0.82	96.0 ± 1.09	88.3 ± 6.5*
Pulmonary shunt (per cent)	0	0.5 ± 1.0%	15.0 ± 1.1%*
V (c.c./min.)	5641 ± 601	5843 ± 860	8405 ± 2850*
f (Respirations/min.)	12 ± 1.82	13 ± 3.6	22 ± 9.4*
V_T (c.c.)	499 ± 58	470 ± 105	384 ± 61.1*
V_D (c.c.)	144 ± 32	152.7 ± 30.2	161.5 ± 51.0
$\dot{V}_D$ (c.c./min.)	1605 ± 421	1712 ± 543	3743 ± 2200*
V_A (c.c.)	310 ± 47.4	271.4 ± 87.9	176.0 ± 46.0*
$\dot{V}_{CO_2}$ (c.c./min.)	212.3 ± 23	203 ± 20.9	237 ± 36.3*
$\dot{V}_A$ (c.c./min.)	3496 ± 381	3261 ± 459	3695 ± 1149*
Vital capacity c.c./M^2	2290 ± 274	2210 ± 290	
Residual volume c.c./M^2	680 ± 148	934 ± 165†	
Reserve volume c.c./M^2	462 ± 116	510 ± 138	
Total lung volume c.c./M^2	2970 ± 313	3150 ± 283	
RV/TLV per cent	23.0 ± 4.1	29.9 ± 5.1†	
Index pulmonary mixing (per cent N_2)	1.45 ± 0.23	2.53 ± 0.85†	
Maximum breathing capacity L./min./M^2	60.7 ± 5.7	48.8 ± 7.3†	

*Statistical analysis reveals this mean to differ significantly from the preoperative mean ($p = 0.05$ or less).

†Statistical analysis reveals this mean to differ significantly from the mean value obtained in 16 normal subjects ($p = 0.05$ or less).

The resting minute volume of ventilation and basal metabolic rates did not differ significantly from the normal values. The arterial blood hemoglobin saturation, carbon dioxide content, carbon dioxide tension, and pH were also

within normal limits. The mean for the oxygen dissolved in the arterial blood by breathing 100 per cent oxygen was not significantly different from the mean value found in normal subjects. From these data the portion of blood being shunted through nonaerated pulmonary tissue was found to be normal preoperatively. This is consistent with the findings reported recently by Ebert.[5]

RESULTS

The mean results of the pre- and postoperative studies are recorded in Table I, and the changes illustrated in Figs. 1 and 2.

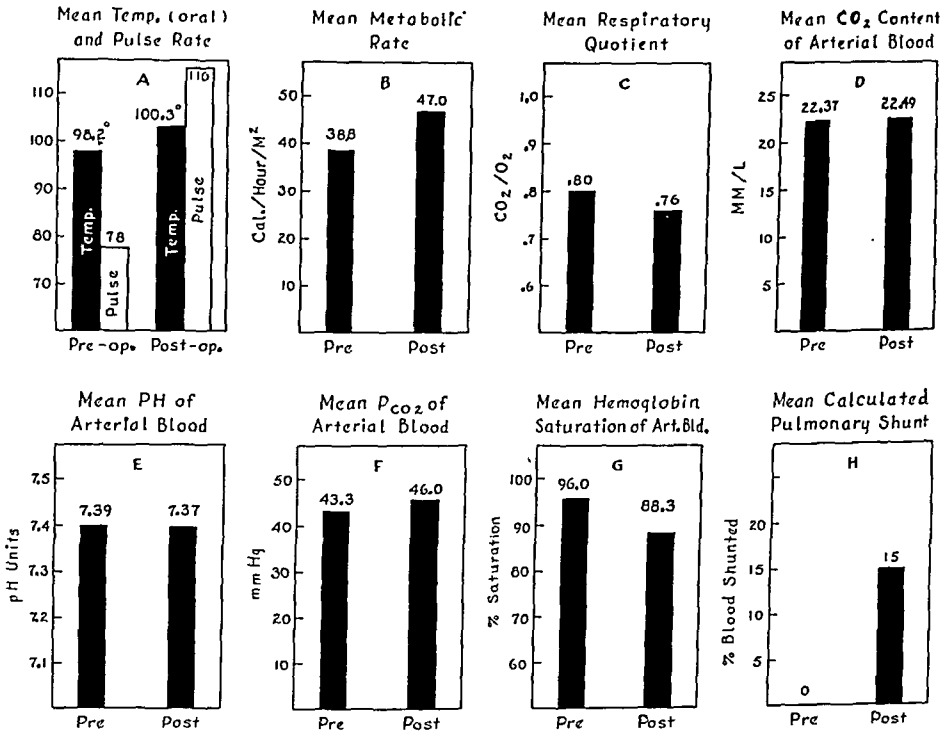


Fig. 1.—Physiologic changes following lobectomy-thoracoplasty (sixteen to twenty hours after surgery). Comparison of the preoperative and postoperative means.

The mean oral morning temperature rose from 98.2° to 100.3° F. the day after surgery (Fig. 1, *A*). The mean preoperative pulse rate was 78 per minute, as compared with a mean of 116 per minute postoperatively (Fig. 1, *A*). The mean postoperative metabolic rate was 38.8 cal./hr./M² preoperatively and 47.0 cal./hr./M² postoperatively (Fig. 1, *B*). The above three changes were all statistically significant. The respiratory quotient remained essentially unchanged pre- and postoperatively (Fig. 1, *C*).

The mean carbon dioxide content of arterial blood was 22.37 mm./L. preoperatively and 22.49 mm./L. postoperatively (Fig. 1, *D*). The pH of the arterial blood was 7.39 before surgery and 7.37 on the morning following the operation (Fig. 1, *E*). The mean P_{CO_2} was 43 mm. Hg preoperatively and 45 mm. Hg postoperatively. None of these changes proved to be of significance.

The mean arterial hemoglobin saturation while the patient was breathing ambient air fell from 96.0 per cent preoperatively to 88.3 per cent postoperatively (Fig. 1, *G*). This change was of statistical significance.

The quantity of oxygen physically dissolved in the arterial blood by exposure to an alveolar oxygen tension of about 600 mm. Hg by the breathing of 100 per cent oxygen fell from 1.99 volume per cent preoperatively to 1.22 volume per cent postoperatively. From this fact one can calculate that a 0.77 volume per cent fall occurred in the oxygen content of the blood leaving the lungs. Such a fall was probably a result of admixture of blood passing through nonaerated lung tissue. This change represents a 15 per cent functional pulmonary shunt (Fig. 1, *H*). This degree of postoperative pulmonary shunt would account for about one-half of the unsaturation of arterial blood which was noted when the patient breathed ambient air.

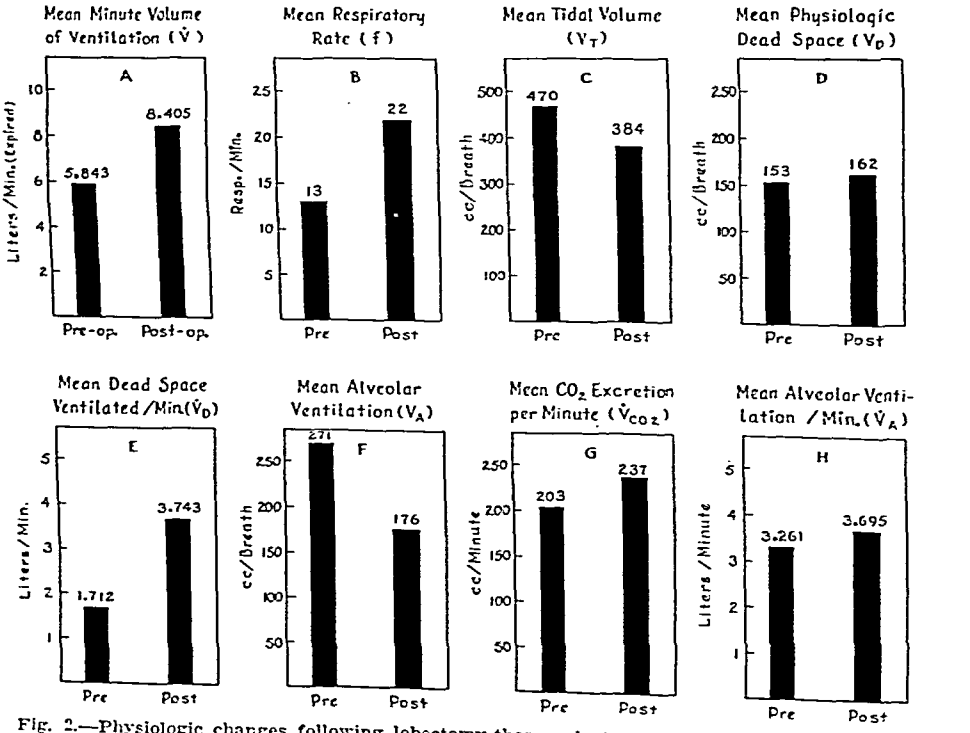


Fig. 2.—Physiologic changes following lobectomy-thoracoplasty (sixteen to twenty hours after surgery). Comparison of the preoperative and postoperative means.

The minute volume of ventilation rose from a preoperative mean of 5,843 c.c./min. to 8,405 c.c./min. on the morning following surgery (Fig. 2, *A*). This change was accounted for largely by an increased rate of respiration from 13.0 to 22.0 per minute (Fig. 2, *B*). The tidal volume fell from a mean of 470 c.c. preoperatively to a mean of 384 c.c. postoperatively (Fig. 2, *C*). These three changes were all highly significant. The volume of physiologic dead space rose significantly (153 c.c. preoperatively and 162 c.c. postoperatively) (Fig. 2, *D*).

The volume of dead space ventilated each minute (physiologic dead space times the respiratory rate) rose from a mean of 1,712 c.c./min. preoperatively to 3,743 c.c./min. postoperatively, a mean rise of 92 per cent (Fig. 2, *E*). This change was highly significant. Alveolar ventilation per breath decreased significantly from 271 c.c. before surgery to 176 c.c. shortly thereafter (Fig. 2, *F*). The rate of carbon dioxide excretion by the lungs rose from a mean of 203 c.c./min. preoperatively to 237 c.c./min. postoperatively (Fig. 2, *G*). From this the calculated alveolar ventilation per minute was found to be increased significantly from a mean of 3,261 c.c./min. preoperatively to 3,695 c.c./min. on the morning after surgery (Fig. 2, *H*). Both of these alterations were statistically significant.

Comparison of These Effects With Those of Thoracoplasty.—The mean physiologic effects of upper lobectomy in combination with a five-rib thoracoplasty were compared statistically with the mean effects rendered by each stage of a seven-rib thoracoplasty (reported in the first paper of this series). Thoracoplasty was used for comparison because its clinical effects are so well known that it can serve as a standard by which the effects of other surgical procedures can be judged. A lapse of sixteen to twenty hours was allowed after surgery before making the postoperative observations in order to allow for the compensation of the effects of general anesthesia (pentothal-curare) used in the

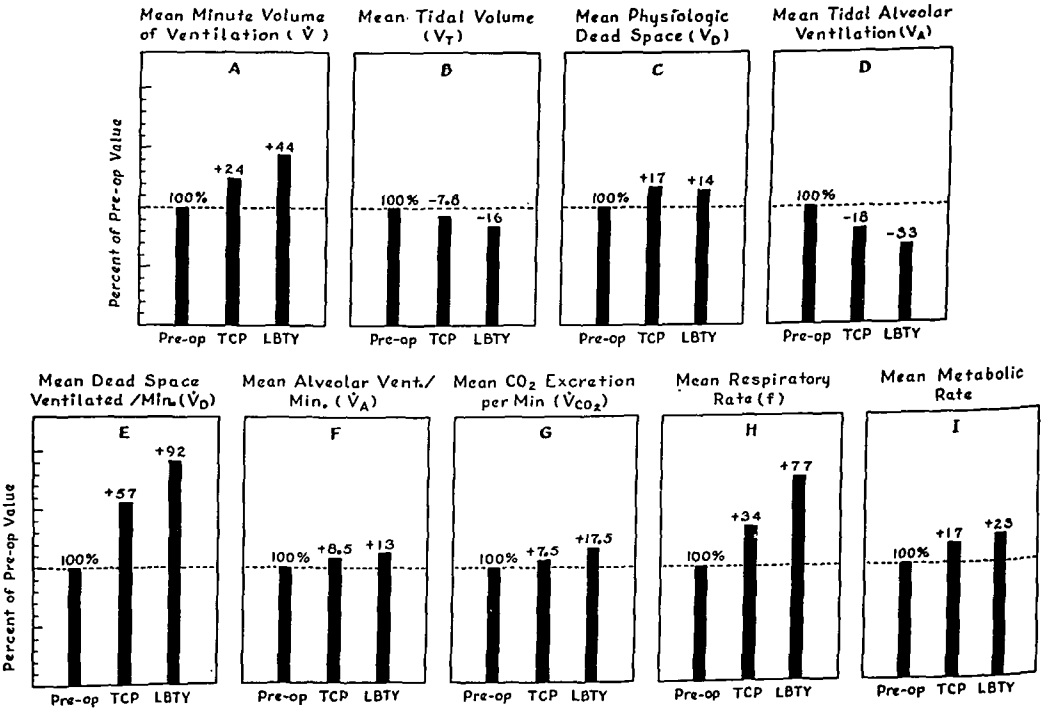


Fig. 3.—Comparison of the physiologic changes following lobectomy-thoracoplasty with those following thoracoplasty alone. The first bar represents 100 per cent of the preoperative value for each patient individually. The second bar represents the weighted mean percentage change of the three postoperative determinations in ten patients undergoing a three-stage, seven-rib thoracoplasty.

present series of patients, since only local anesthesia was used in the thoracoplasty series. The data collected after such compensation should be comparable to those obtained after thoracoplasty. The means of the percentage change from the individual preoperative values were compared for the two groups by the use of the t test. The most significant data are presented in Fig. 3 in which the postoperative changes following the two procedures are compared with the mean preoperative values as 100 per cent.

The increase of 44 per cent in the respiratory minute volume following lobectomy-thoracoplasty was significantly greater than the increase of 24 per cent following each stage of thoracoplasty alone (Fig. 3, *A*). Other measurements which showed a statistically greater alteration in the present series over those found in thoracoplasty were: (1) a fall of 16 per cent in tidal volume (Fig. 3, *B*); (2) a fall of 33 per cent in tidal alveolar ventilation (Fig. 3, *D*); (3) a rise of 92 per cent in volume of dead space ventilated per minute (Fig. 3, *E*); (4) a rise of 13 per cent in minute alveolar ventilation (Fig. 3, *F*) associated with an increase of 17.5 per cent in the elimination of carbon dioxide (Fig. 3, *G*); (5) a rise of 77 per cent in respiratory rate (Fig. 3, *H*); and, (6) a rise of 28 per cent in metabolic rate (Fig. 3, *I*). There was a small rise in physiologic dead space (Fig. 3, *C*) (14 per cent) which was similar to the change observed after each stage of thoracoplasty. This is probably attributable to the increased rate of respiration.

The rise in the temperature and pulse rate was significantly greater in this series of patients than in the patients undergoing thoracoplasty. Carbon dioxide content, pH, and carbon dioxide tension of the arterial blood were not altered significantly in either series sixteen to twenty hours after completion of the surgery. This fact gives evidence that the effects of general anesthesia had been compensated since it has been shown that respiratory acidosis due to CO_2 retention occurs during thoracic surgery performed under general anesthesia.

These data indicate that the lobectomy-thoracoplasty combination was of significantly greater magnitude than one stage of a thoracoplasty.

CASE REPORT

One death occurred in the present series, and it seems worth while to consider the data obtained in detail. The patient, H. R., was a white man aged 41 years. *History:* Bilateral upper lobe infiltrate was first discovered on separation roentgenogram in 1944. Sputum was found to be positive for tubercle bacilli. He was asymptomatic except for mild ease of fatigue. He was hospitalized and treated with bed rest from June, 1944, to June, 1946, when an artificial pneumothorax was induced on the left. There were no complications with the induction, but pleural fluid began to accumulate after two years. The sputum became negative thereafter, and he was discharged from the hospital in November, 1947. Pneumothorax was continued on an outpatient basis, and he returned to school. In October, 1949, there was a recurrence of ease of fatigue accompanied by pain in the right side of the chest. He was admitted to this hospital at that time. Several gastric washings had been positive since 1947. *Physical Examination:* He was well developed and well nourished, and was in no distress. Representative roentgenograms are shown in Fig. 4. At the time of surgery there was evidence of a bilateral nodular infiltrate and some pleural thickening on the left. No pneumothorax remained on the left. Planigrams of the right lung revealed multinodular infiltration with cavitation. The left side showed pleural thickening. The vital capacity

in February, 1951, was 4,200 c.c. and was rapidly expelled. Differential bronchospirometry revealed that the left lung accounted for 35 per cent of the total ventilation and oxygen absorption. The patient was treated with bed rest from October, 1949, to March, 1951. No chemotherapy was used during this period.

On March 19, 1951, resection of the right upper lobe was attempted. Because of extensive tuberculous involvement of the middle lobe, it was also sacrificed, and a five-rib posterior thoracoplasty was performed to close the dead space. The patient was dyspneic from the time of the surgery, and the respiratory rate ranged from 40 to 48 per minute. Oxygen therapy gave little or no relief of the dyspnea, but did relieve the cyanosis. On March 22, 1951, there was a sudden onset of auricular fibrillation, but the normal sinus rhythm was restored by the intravenous administration of Cedalanid. He showed occasional episodes of disorientation. He became moderately cyanotic when breathing room air. Drainage catheters were removed on the fourth postoperative day, and the wound was noted to be healing

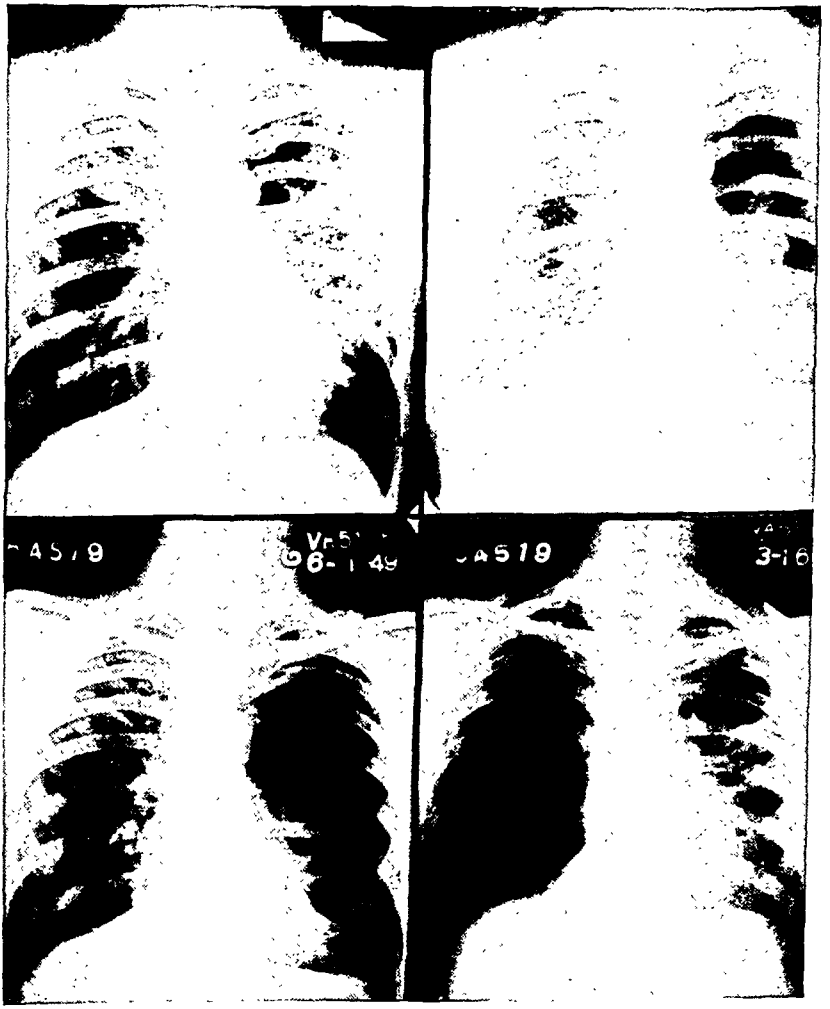


Fig. 4.—Case H. R. *Upper left,* film taken in November, 1944; *upper right,* film taken in March, 1947; *lower left,* June, 1949; and *lower right* shortly before surgery in March, 1951. The left pneumothorax is seen in the 1949 film, and the pleural residuals are brought out in the film of March, 1951. The nodular disease in the right upper and middle lobes was better demonstrated by planigraphy.

normally. Dyspnea continued to be so severe that simple conversation caused fatigue. On the evening of March 28, 1951, he received visitors for ninety minutes and seemed cheerful, in spite of the dyspnea. As the visitors left he commented to the nurse that he felt "all tired out." Ten minutes later he was found unconscious with very weak gasping respirations and a dusky gray cyanosis. He expired within a few minutes. The pulse was noted to be present after he had lost consciousness, but there was no mention of its character.

TABLE II. CASE H. R.—44 W. M.

	PREOPERATIVE	POSTOPERATIVE	PER CENT CHANGE
Oral temperature (°F.)	97.80	101.20	+3.5
Pulse rate per minute	82.00	126.00	*+53.7*
Metabolism rate (Cal./hr./M²)	34.70	48.60	*+40.1*
Respiratory quotient	0.80	0.76	−5.0
Arterial blood: pH	7.39	7.34	−0.7
CO_2 content mm./L.	24.28	20.79	−14.4
P_{CO_2} (mm. Hg)	47.00	45.00	−4.2
Hemoglobin saturation (per cent)	94.20	84.9	−9.9
Pulmonary shunt (per cent)	0.00	11.90	
$\dot{V}$ (c.c./min.)	6,045.00	14,270.00	*+135.4*
f (resp./min.)	15.00	46.00	*+206.0*
V_T (c.c.)	403.00	311.00	*−22.8*
V_D (c.c.)	150.20	171.20	*+14.0*
$\dot{V}_D$ (c.c./min.)	2,253.00	7,875.00	*+249.5*
V_A (c.c.)	206.80	93.80	*−113.0*
$\dot{V}_{CO_2}$ (c.c./min.)	20.90	281.00	*+34.4*
$\dot{V}_A$ (c.c./min.)	3,101.00	4,313.00	*+39.1*
Vital capacity c.c./M²	1,759.00		
Residual volume c.c./M²	963.00		
Reserve volume c.c./M²	280.00		
Total lung volume c.c./M²	2,722.00		
RV/TLV per cent	35.40		
Index pulmonary mixing (per cent N_2)	4.25%		
Maximum breathing capacity L./min./M²	40.62		
Surface area (M²)	2.16		

Physiologic measurements were obtained before surgery and sixteen hours after the completion of the eight-hour surgical procedure. These data are given in Table II. It can be seen that the arterial blood showed no dangerous abnormality at the time of the postoperative study (sixteen hours after surgery). The amount of carbon dioxide excreted was increased in association with a slight fever. This stimulated an increased alveolar ventilation. The tidal volume was limited to 311 c.c. instead of the preoperative value of 403 c.c. In association with these alterations there was a rise in the respiratory rate from 15 to 46 per minute, which resulted in maintenance of adequate alveolar ventilation. This increased respiratory rate entailed an increase in dead space ventilation from 2,253 c.c. per minute to 7,875 c.c. per minute. The great burden which this situation constituted was not fully appreciated at the time. The patient died rather suddenly after talking with visitors. Autopsy failed to reveal a definite cause of death. The remaining lung tissue showed no congestion or edema to indicate heart failure, and the heart was normal. There was no clot in the pulmonary

artery. The pleura over the left lung was thickened, and the underlying left lung contained many caseous nodules which proved to be typical of tuberculosis on section. The presumption is that the crippled and encapsulated left lung was barely able to carry the respiratory load postoperatively. The tidal volume was limited not only by the pain of the operated (right) side, but also by the pleural residuals on the left. We postulate that the diaphragm eventually became so fatigued that it would no longer respond with rhythmic contractions. It is felt that the increased production of carbon dioxide coupled with some crippling of the contralateral lung, the limitation of the tidal exchange, and a rather significant degree of paradoxical respiration associated with the five-rib thoracoplasty all contributed to the death. It is quite possible that a decrease in the volume of physiologic dead space (i.e., tracheotomy) might have allowed adequate alveolar ventilation to be accomplished with a great deal less effort. This is postulated because of the fact that the greatest abnormality found in the physiologic data was in the tremendous increase in the ventilation of the dead space.

DISCUSSION

Resectional surgery as a primary approach to the therapy of pulmonary tuberculosis is still in the developmental stage. The magnitude of the resection may vary from the removal of a small caseous nodule to a complete pneumonectomy. A great deal of work is currently being done in evaluating the efficacy of the various procedures as tools in overcoming tuberculosis. The present work is an attempt to determine the physiologic magnitude of each procedure, in order to aid in the selection of patients who are suitable for the various procedures. No consideration has been given to the therapeutic efficacy of the operations.

The most important principle involved in the modern surgical approach to pulmonary tuberculosis seems to be the extirpation of the significant disease without overexpansion of the remaining pulmonary tissue. Where the volume of tissue removed is small the problem of overexpansion is probably not important. But where as much as a lobe or major part thereof is to be removed, many surgeons and internists alike feel that there is an advantage in lessening the space which must be filled by the remaining lung tissue.

It was for this reason that a one-stage lobectomy and five-rib thoracoplasty was tried somewhat as a short cut to avoid two separate procedures.[2-4] The present study was undertaken in order to define the physiologic magnitude of this type of surgery and to compare it with the familiar effects of thoracoplasty.

All procedures which involve the removal of several ribs have one thing in common: i.e., the presence of a flail chest wall. The first paper in this series attempted to point out the disadvantage at which thoracoplasty places the respiratory mechanism. It was shown that tidal volume is limited immediately postoperatively (probably by pain and by the presence of paradoxical respiration). This necessitates an increased respiratory rate if alveolar ventilation is to be maintained. The maintenance of even the same rate of excretion of carbon dioxide is then more difficult than before surgery. The disadvantage against which the patient is working may be accentuated by the need for an increased rate of carbon dioxide elimination, such as occurs (1) with the acidosis which develops during general anesthesia, and (2) with postoperative fever.

The data herein presented reveal the physiologic magnitude of the combination of lobectomy and five-rib thoracoplasty to be significantly greater than

that of one stage of a seven-rib thoracoplasty. The increases in the metabolic rate, postoperative temperature, pulse rate, and carbon dioxide excretion were all significantly greater in the present series than in any stage of thoracoplasty alone. That the body labors under the same type of disadvantage following both procedures is shown by the limitation of tidal volume and tidal alveolar ventilation, and the great increase in respiratory rate and in dead-space ventilation.

The practical importance of these observations is obvious. It seems clear that the selection of patients who are suitable for the combination of resection and thoracoplasty must be rather carefully made. The mechanical and physiologic disadvantage at which the body is placed is very similar for the two procedures, probably because both involve the removal of several ribs with the resultant loss of respiratory efficiency. However, the magnitude of the effects associated with combined lobectomy-thoracoplasty is significantly greater than those associated with thoracoplasty alone, probably because of the greater surgical trauma associated with pulmonary resection per se.

It should be emphasized that the observations reported here were made after the subjects had accomplished complete correction of the respiratory acidosis which commonly occurs during thoracic surgical procedures. The need for eliminating the extra carbon dioxide which is frequently retained during general anesthesia may temporarily impose a very heavy load upon the crippled respiratory mechanism. An increase in the rate of respiration constitutes the only means by which the body can accomplish sufficient ventilation to achieve the elimination of any extra carbon dioxide. It is obvious that a patient with a greatly limited maximum breathing capacity and exercise tolerance before surgery might be unable to increase the rate and volume of ventilation sufficiently to meet the demands placed upon the respiratory system in the postoperative period. In the case of H. R. (reported above) there was a combination of pulmonary insufficiency, lung encasement, and paradoxical chest motion. It was only by great effort that he was able to maintain a passable alveolar ventilation. Finally, after the mild exertion of talking with visitors, the muscles responsible for carrying the extra work of respiration became fatigued and respiration ceased.

SUMMARY AND CONCLUSIONS

1. Eleven male patients undergoing an upper lobectomy in combination with a five-rib thoracoplasty were studied in the manner previously reported from this laboratory.

2. The inefficiency of the respiratory mechanism following lobectomy-thoracoplasty was significantly greater than that found immediately following each stage of thoracoplasty alone. Also, the magnitude of the stress placed upon the body by the combination of lobectomy and thoracoplasty was greater than that imposed by each stage of thoracoplasty alone.

3. There was one postoperative death in the present series, and this case is described in detail. The most significant features of the case were the great increase in the respiratory rate and in the dead-space ventilation and the de-

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crease in tidal exchange. Autopsy revealed no cause of death, and it is concluded that the physiologic alterations found probably afford an adequate explanation for this postoperative death.

4. It is concluded, finally, that only patients with good respiratory reserve should be selected for a combination of thoracoplasty (which greatly cripples the efficiency of the respiratory mechanism) and pulmonary resection (which imposes a considerably greater load upon the body than does thoracoplasty alone).

The author is indebted to Dr. R. V. Ebert for much helpful advice, and to Shirley Lundgren and John Oganovich for technical assistance.

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THE INTRATHORACIC USE OF THE LARGE BOWEL AFTER SUBTOTAL ESOPHAGECTOMY FOR CANCER

Report of One Case

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São Paulo, Brazil

THE surgical management of lesions of the esophagus has improved considerably in the last fifteen years because of advances in the field of modern anesthesia and the discovery of new antibiotic drugs. However, the reconstruction of the organ after resection, or its complete substitution when necessary, is a problem still challenging the skillfulness of all those who have tried to solve it.

The multiplicity of approaches to the esophagus, the variety of methods used, and the continuous appearances of new publications on the subject show that esophagoplasty has not yet come to its final stage of development, and that something is still unconquered by the surgeon. In reviewing these techniques we felt that perhaps the utilization of a segment of transverse and descending colons could give us a better chance to complete the operation begun by the esophagectomy, restoring the continuity to this part of the alimentary canal.

CASE REPORT

J. D. I. was a 51-year-old, white, married, Portuguese man, a baker. He came to the surgical clinic in November, 1950, complaining of retrosternal pain and progressive dysphagia for the last three months. He complained that he could not even drink water during the two days before admission. At the physical examination it seemed that he had lost much weight, but he still looked well. The blood pressure was 130/80. The fluoroscopic examination* revealed an obstruction localized between the upper and the *middle thirds of the esophagus, showing irregular filling at the level of the aortic arch,* with subtotal stenosis. Adequate preoperative care restored the blood cells to normal and the serum proteins to their level; the patient was considered in good condition for an operation. On Dec. 29, 1950, he was operated on under endotracheal general anesthesia (ether-oxygen mixture). Induction was carried out with sodium pentothal. The chest was entered through the left side, the sixth rib being resected in its whole extension (Fig. 1). Also the posterior segment of the fifth rib was resected. On exploring the mediastinum a tumor was identified in the esophagus at the level of the aortic arch. Further examination revealed this tumor spreading all over the organ to the diaphragm, but it did not invade the periesophageal tissues, thus permitting the dissection of the organ from the surrounding structures. Firm adhesions fixed the aorta and the esophagus at the level of the aortic arch. The diaphragm was then opened from its esophageal hiatus to the anterior abdominal wall and exploration was performed for metastasis in the liver and lymphatic nodes. As these structures were free from metastasis the stomach was sectioned from the esophagus just below the cardia and this opening closed with inverting cotton sutures in two planes. The lower end was closed with cotton in a purse-string fashion and the esophagus was easily isolated from the mediastinum to the aortic arch. At this point the adhesion was very firm, but it also came away. Palpation conducted over this area revealed that the tumor spread upward making it necessary to dissect the organ high into the neck.

From The Surgical Clinic Department, Instituto dos Comerciarios São Paulo.
Received for publication June 20, 1952.
*Performed by Dr. F. Bevilacqua.

As the patient was in a very poor general condition at this moment and the stomach was very small and retracted, it was felt that it would be difficult to bring it up to an anastomosis with the proximal end of the esophagus in the neck. As the dissection of the esophagus was completed up to the neck, it was left in the mediastinum, its lower end covered by a rubber finger, and the thorax was closed with nonabsorbable suture material by planes. The patient was then placed on his back and a gastrostomy of the Witzel type immediately performed; following an incision on the anterior border of the left sternocleidomastoid muscle the total thoracic esophagus was brought up to the neck and exteriorized (Fig. 1). The cervical esophagus also presented tumorlike nodules and for this reason it was resected, leaving only the first 3 or 4 cm. of the organ which were free of tumor masses. The proximal end of the organ was implanted in the skin of the neck where it formed a fistula. It must be emphasized that an attempt was made to remove all the visible periesophageal lymph nodes together with the organ while performing this subtotal esophagectomy for generalized cancer.



Fig. 1.—Patient showing incisions in the neck and chest; the cervical and epigastric fistulas.

A grayish fragment of this tumor tissue from the wall of the organ as well as a black lymph node were sent for pathologic examination.

Microscopically, the first of them showed an immature neoplasm of epithelial cells whose elements were grouped in irregular blocks exhibiting great polymorphism, as well as typical and atypical mitosis. The second material showed the structure of lymphatic nodular tissue substituted by connective proliferation presenting hyalinized points filled by anthracotic pigment and negative images of crystals together with small areas of neoplastic blocks of the type described. *Diagnosis** was squamous carcinoma of the esophageal wall, carcinomatous metastasis in the lymph node, anthracosilicosis of the lymphatic gland.

*By Dr. Walter E. Maffei.

The early postoperative period was stormy, but in a few days the patient presented rapid and evident improvement in general condition. Fifteen days after the surgical procedure he had already gained some weight and three months later he had gained 13.2 pounds.

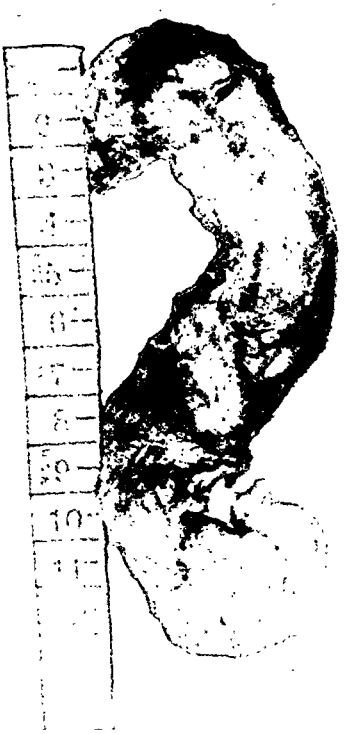


Fig. 2.

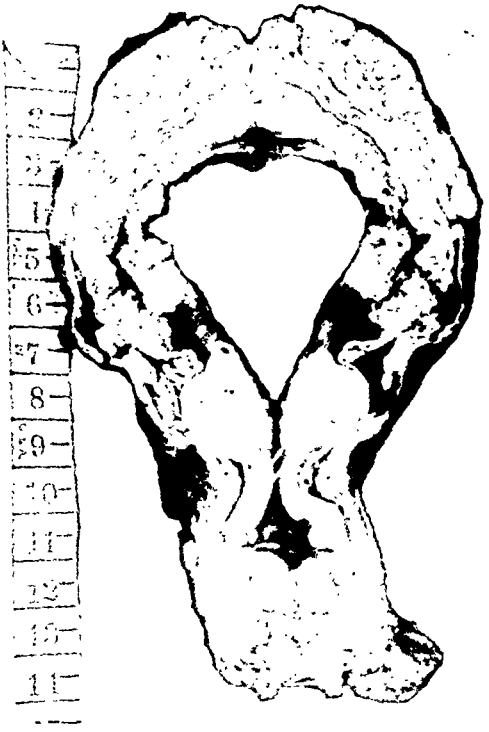


Fig. 3.

Fig. 2.—Superficial aspect of the entire esophagus.

Fig. 3.—The esophagus opened through a longitudinal section showing the tumor along its surface.

Unfortunately the idea of eating by mouth had grown more and more in the patient and finally forced us to perform the second stage three months after the first stage, and therefore three months before the time was up, which had been hoped for as an observation period for possible metastasis. In March 31, 1951, after a convenient check-up of his condition, a reconstruction of the passage was attempted. In a right dorsal lateral position (45 degrees more or less) and under the same anesthetic mixture the patient was again operated on. Starting at the left paravertebral region an incision was made resecting the scar of the previous operation extending to the costal margin which was sectioned. This same incision was then extended downward to the abdominal anterior wall and parallel to the internal border of the left rectus muscle to the extent of three fingers below the umbilicus (Fig. 4). Both thoracic and abdominal cavities were opened, the diaphragm being sectioned in the same direction as before. It was decided to leave the feeble adhesions between the left lung and the thoracic wall to be freed just after the accomplishment of the abdominal stage.

On exploring the peritoneal cavity a retracted stomach was found, strongly adherent by the previous gastrostomy to the anterior abdominal wall and also to the gastrohepatic ligament. There were found in this last structure some hard and grayish masses, which

were dissected and proved to be carcinomatous metastases. There were no suspected nodules in the mediastinum.

The great omentum was then isolated from the transverse colon and its connections to the splenic angle. Afterward the transverse and descending mesocolons were explored and their arterial circulation studied carefully. The middle colic artery was easily identified by its diameter and preserved. All other branches to the left were ligated to its inosculation to the first sigmoid artery, which also was preserved. The next step was the dissection of the transverse and descending mesocolons in their entirety. In this dissection care was taken to preserve the middle colic artery and all its branches together with the isolated loop that we wanted to free, maintaining in this way its blood supply. By this procedure we got a long and movable colonic segment attached to the abdomen by only a long mesocolic pedicle containing the middle colic artery (Fig. 5). This pedicle reached the isolated colonic segment near its proximal extremity (that is, the right end of the isolated loop).

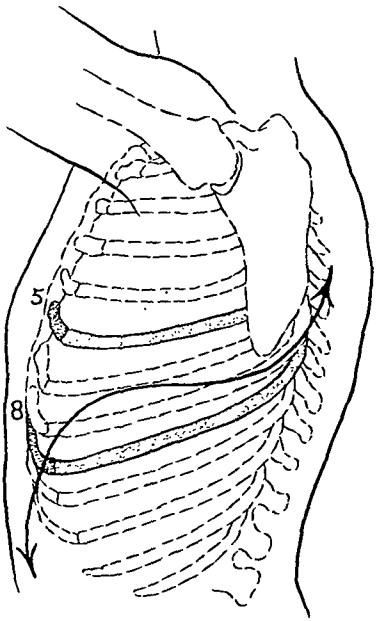


Fig. 4.

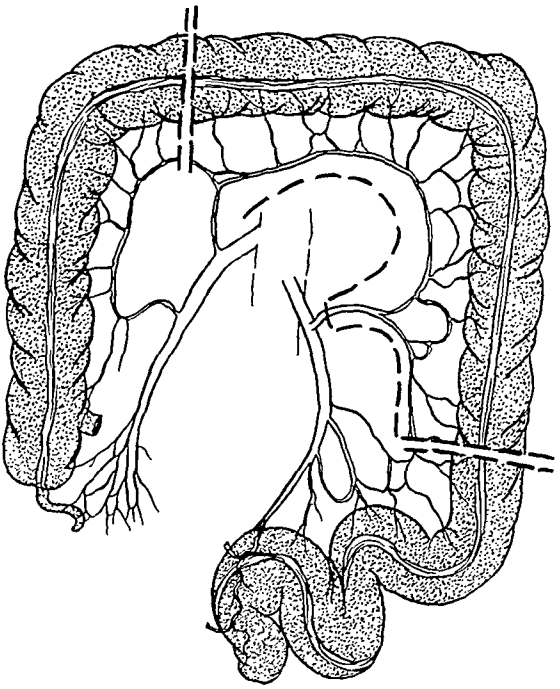


Fig. 5.

Fig. 4.—Line of incision utilized in the attempt to perform an intrathoracic esophagoplasty.

Fig. 5.—Diagrammatic sketch showing the segments of isolated colon used in the chest and the circulation used.

The colonic continuity was re-established by an end-to-end continued suture with chromic catgut, using the Vasconcellos clamp. A second plane using cotton stitches was done over the first one. The opening in the mesocolon was closed completely, then the proximal end of the isolated colonic segment was anastomosed to a new opening made at the highest point of the gastric wall by continuous chromic catgut suture in two planes. Careful attention was given to the arterial mesocolic pedicle which occupied a better position posteriorly to the stomach where it was not exposed to any traction. Also care was taken not to twist this isolated loop to the stomach. After his cologastric anastomosis was ready, the stomach was brought up as far as possible to the esophageal hiatus of the diaphragm now wide open and fixed to the borders of this muscle, with the intention of

preventing any traction on the cologastric suture. This maneuver permitted a small portion of the stomach, containing cologastric anastomosis, to remain supradiaphragmatically. Sufficient room was left between the stomach and the border of the diaphragm for passage without constriction of the arterial pedicle going to the isolated segment. In order to avoid any possible damage to this pedicle, a splenectomy was carried out. The left pleural space was then freed of its adherence to the left lung and the isolated segment was conducted and placed in a good position to the internal side of the posterior thoracic wall near the mediastinal aspect, where it was fixed by anchoring stitches. The diaphragm was then completely closed to the most anterior portion. The abdomen was also closed in planes using cotton interrupted suture.

A left cervical incision over the anterior scar was made and all the elements were incised and retracted until a plane could be reached posterior to the left clavicle. Through the left transthoracic opening a connection was tried between the cervical region and the highest part of the left thoracic cavity, close to the middle line. During this procedure a large retroclavicular venous blood vessel was severed, producing a hemorrhage which was controlled with great difficulty. As the patient entered hemorrhagic shock we were obliged to alter plans for this intervention, and instead of making the cervical anastomosis between the esophagus and the colon at this stage we decided to implant the colon to the anterior thoracic wall. The distal end of the isolated colonic loop was fistulized to an opening made in the left, second intercostal space, two fingers from the sternal border. It was sutured by interrupted cotton stitches in two planes, one muscular and the other cutaneous. Penicillin and streptomycin were placed in the left cavity which was closed by planes without drainage. The left cervical incision was then closed by interrupted cotton sutures.

In summary, an attempt was made to perform an intrathoracic reconstruction of the esophagogastric continuity by using antiperistaltically a segment of transverse and descending colons after a subtotal esophagectomy for carcinomatosis of the esophagus (Figs. 2 and 3).

Even though this patient was able to re-establish his blood pressure seven hours after the operation a new hemorrhage at the same point brought him again into shock. Treated correctly his blood pressure returned to its normal level. However, forty-eight hours after the intervention there was a very serious overflow of saliva and other secretions to the tracheobronchial tree. Two bronchoscopic aspirations were necessary to restore his general condition to normal once more. At the end of the third postoperative day he had the first spontaneous bowel movement, with a second one during the night.

Also the intrathoracic loop started to move, beginning by eliminating fecaloid fluid material and later gastric contents. By introducing a tube into the chest opening, this loop could be washed of its contents with saline solution. After this procedure it was noted that any fluid introduced into the loop could be seen appearing spontaneously through the gastrostomy some time later demonstrating that this loop was functioning toward the stomach.

The general condition of this patient improved rapidly, and he reported he felt much better. In the afternoon of the eighth postoperative day following a severe cough, there was dehiscence of the abdominal part of the surgical wound, through which jejunal loops were protruding. Developing shock, he was again taken to the operating table and under general anesthesia all the protruding jejunal loops were put back into the peritoneal cavity. The examination of the peritoneal cavity made at this moment did not reveal abnormality or any sign of dehiscence of the colocolic anastomosis whatever. However, a certain amount of serofibrinous exudate was found in the cavity and was attributed to the prolonged evisceration of the loops. Penicillin and streptomycin were placed in the peritoneal cavity before it was closed by separated cotton stitches. Once more the patient came out of shock and there was an improvement in his general condition. All the thoracic symptoms and signs indicated a better local condition. This was confirmed by several x-ray pictures taken of the patient in bed during this postoperative period.

Suddenly on the night of the eleventh day he felt ill and died. The autopsy report, performed at the Institute Adolpho Lutz, presented the following main points.

General Data.—A white, adult man presented three cutaneous fistulas localized as follows: The first, on the anterior border of the sternocleidomastoid muscle, two fingers above the internal end of the left clavicle; the second, two fingers from the left sternal border, in the second interspace; the third in the epigastric region, in the middle line, where a rubber tube can be seen. Three surgical incisions were noted: The first corresponding to the first fistula along the muscle; the second already healed in the middle line, xipho-umbilical, where there was a third fistula. The third surgical scar was recent and irregular, going from the left paravertebral space to the anterior aspect of the chest forming an arch over the left sixth rib, to the costal margin and downward over the anterior rectus muscle to a point inferior to the umbilicus. This last incision presented cotton stitches over its line. By opening this incision it was noticed that the sixth rib had been removed surgically. Both chest and abdominal cavities were then open and only separated by the diaphragm. In the abdomen it was noticed that a serofibrinous exudate extended over the cavity. In the left side of the abdomen there was a fecaloid material exuding from a dehiscence in the line of suture of the end-to-end anastomosis between the right part of the transverse colon and the sigmoid colon. It was still noted that the stomach was attached to the anterior abdominal wall, where there was a gastrostomy. The highest part of the stomach was sutured and surrounded by the diaphragm entering into the thorax to some degree. By cutting the stitches on the diaphragm it was observed that a pedicle of the mesocolon entered the chest cavity behind the stomach. On opening the diaphragm, a dark fluid was emitted from the left pleural cavity to the abdomen. The transverse and descending colons were in the left pleural cavity extending from the second, left interspace to the highest part of the stomach. Both the superior colonic extremity as well as the cologastric anastomosis showed good healing. There was no spleen (removed).

Diagnosis.—Generalized peritonitis due to dehiscence of anastomatic line of the end-to-end colocolic suture. Gastrostomy (operation). Colostomy in the second left interspace (operation). Supradiaphragmatic cologastrostomy in the left pleural space (operation). Fistula of the esophagus on the left side at the base of the neck (operation). Splenectomy (operation).

Description of Organs Involved.—Esophagus: It is noted that the esophagus was turned toward the cutaneous fistula in the neck and that the esophagus was reduced to only 3 or 4 cm. of its upper third. Thoracic cavity: The cardiac area was normal; pleural membranes were free. The left pleural space exhibited approximately 500 Gm. of dark fluid. Right lung: Form and size were normal. Crepitation was present. The sagittal section was dark red in color, emitting when compressed a small amount of blood (slight congestion). Left lung: The size was small due to collapse of the lower lobe. Cut surface was normal. Heart: No information worth noting.

Causa Mortis.—Toxemia and generalized peritonitis due to dehiscence of suture line in the intestinal anastomosis.*

COMMENTS

The main objective of this paper is the presentation of a case of generalized carcinoma of the esophagus treated by a subtotal esophagectomy where we had to face the problem of a plastic reconstruction of the organ.

It was clear from the beginning of the surgical treatment that this patient could not expect to have a long survival due to metastasis already observed at the first stage of the operation. However, his will to live and to eat again

*Dr. Pedro Gherardi, Jr.

through his mouth had forced us to break into our observation period three months from the first operation and try a re-establishment of the continuity of the upper digestive tract. Considering the conditions revealed at the time of the esophagectomy, we had no doubts about the unfeasability of bringing his stomach up to the neck, as was our intention at first. Besides its small size, the stomach should be expected to be firmly adherent and fixed to the anterior abdominal wall at the point where the previous gastrostomy was done.

Upon analyzing the advantages and disadvantages of the different methods in a review of the surgical literature and after reading Orsoni's paper[6] a few days before the operation, we decided on an intrathoracic colonic substitution of the esophagus as perhaps the best chance we could give to our patient, even though it was observed that the colon had never been used for this type of plastic surgery until this time. We also were, and still are, favorable to the one-stage resection with an esophagogastric anastomosis described by Sweet, Garlock, and many others, but we are sure this procedure can be useful in cases where the lesions, whatever they may be, are located very high and the stomach cannot be brought to that point.

REVIEW OF LITERATURE ON INTRATHORACIC ESOPHAGOPLASTY

Although prethoracic esophagoplasty has had very satisfactory results, many authors since the beginning of the century have tried to obtain an intrathoracic esophagoplasty. Attempts were made by Wullenstein (1904), Sauerbruch (1905), Willy Meyer (1913), Marwedel (1913), Enderlen and Hotz (1914) and others to perform this intervention on an experimental basis. Lorthioir and his associates tried to establish an anastomosis to the jejunum after the resection of the diseased segment of the esophagus. Kümmel[1] was the first to use an intrathoracic approach by the Kirschner method, doing an anastomosis between the esophagus and the stomach freed from its vascular connections. However, the majority of these interventions were performed in cases of esophageal stenosis due to lye strictures. Tumorous cases were considered very dangerous and useless.

Many tissues and organs have been used up until the present time to substitute for the diseased esophagus, for example, the skin, epidermis, fascia lata, as well as different parts of the digestive tract (stomach, jejunum, and colon).

In 1946, Rienhoff[2] was the first to use jejunum intrathoracically. He made the anastomosis of a free proximal jejunal loop antiperistaltically to the esophagus at the level of the aortic arch (Fig. 6). Bricker, Burford, and Eiseman[3] related in 1949 the re-establishment of the upper thoracic esophagus resected by using a tubed pedicle graft obtained from the cervical regions, which was introduced into the upper thorax and then anastomosed to the lower esophagus Fig. 6).

At the same time, Harrison[4] described the technique used by him in two cases. He re-established through an end-to-end suture the transit of an esophageal segment resected previously using an isolated jejunal loop. This loop was sutured to the esophagus in the neck and below to the gastric fundus completing an isoperistaltic intrathoracic esophagoplasty (Fig. 6).

In November, 1950, Robertson and Sarjeant[5] published a paper on the reconstruction of the esophagus in one stage by using an isoperistaltic segment of the jejunum through an undermined tunnel in the anterior mediastinum (Fig. 6); from the technical surgical point of view, the descriptions of the methods devised by Bricker and associates, Harrison, Robertson, and Sarjeant are excellent and should be read by those considering all these procedures.

In March, 1951, Paul Orsoni presented a study on the utilization of the transverse and descending colons for the intrathoracic plastic reconstruction of

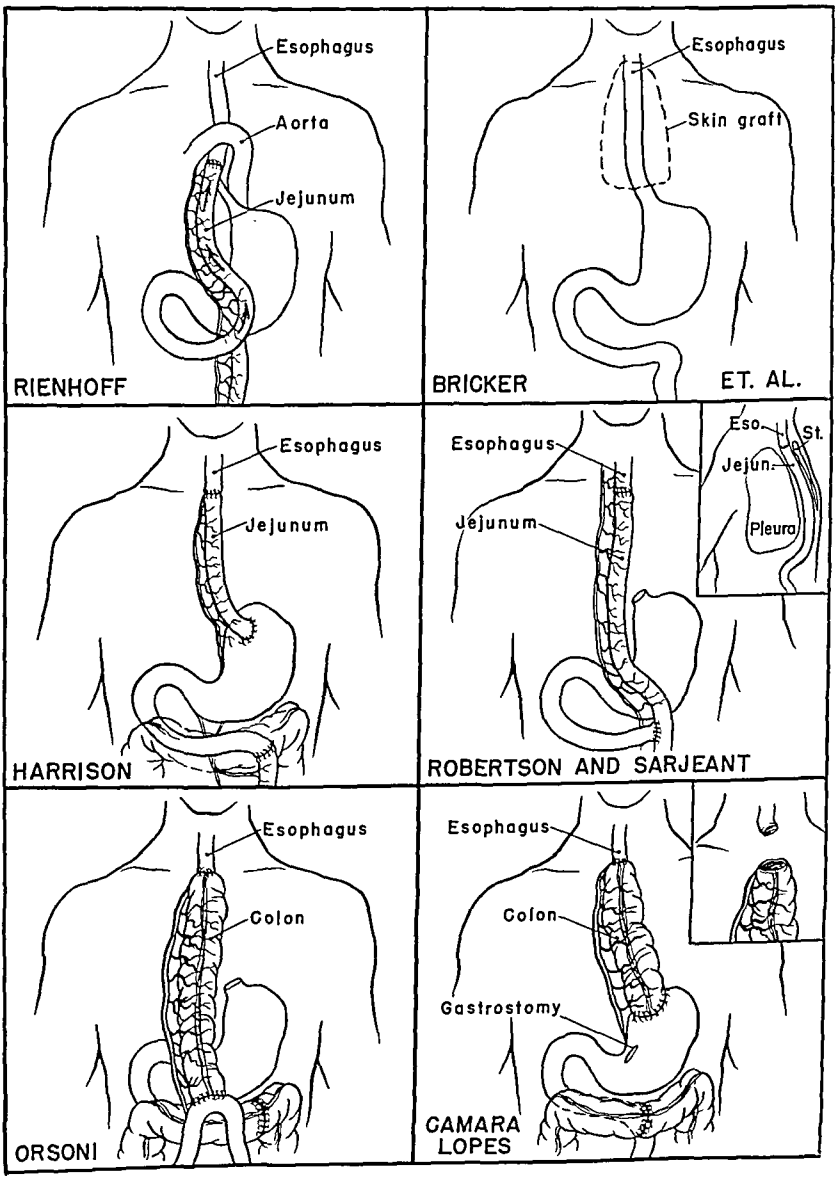


Fig. 6.—The different types of intrathoracic esophagoplasties.

the esophagus. The reasons for the use of this organ were studied by Lemaire, who performed several arteriographies on the colic arteries. Orsoni considered the thoracic use of the colon most advisable. Together with Lemaire (Fig. 6) they have anastomosed an isolated colonic segment between the left portion of the resected esophagus and the first jejunal loop in an attempt to avoid any possible gastric regurgitation to the esophagoplasty so performed.

DISCUSSION

While most of our experience could be looked upon as too limited we feel that this procedure is worth describing and should be kept in mind by others as something else to add to the resources useful to those who have to handle lesions of the upper esophagus.

In reviewing the articles published by different authors on the pre- and intrathoracic esophagoplasty and the results heretofore obtained with them, we have come to the following conclusions.

1. According to the recent works of Toupet and Lemaire, colonic circulation is better than jejunal and can be considered quite sufficient to maintain an isolated colonic segment alive and in good condition, regardless of the length of this segment.

2. Transverse colon can be used iso- or antiperistaltically as has been demonstrated in the cases related by Kelling and Vulliet.

3. According to Reagan and Puestow, working with isolated segments, the colon still keeps its responses to certain drugs, although it loses its mesenteric innervations, a fact that cannot be reproduced in the jejunum.

4. Experiments performed by several authors (Moroney, Flory, Harding, Ivy and co-workers) proved the colon to be more resistant to digestive secretions than jejunum, therefore being much more applicable for its use in esophagoplasty.

5. From the anatomical and functional point of view it is more advisable to perform an esophagoplasty between the upper part of the cervical esophagus or pharynx and the stomach, than to make an anastomosis of its lower extremity to the jejunum as was done by Rienhoff, Robertson and Sarjeant, and Orsoni and Lemaire, keeping in this way the gastric digestive function.

6. The colon is more resistant and less sensitive than jejunum.

7. Much smaller than the stomach in diameter, the colon occupies a smaller space inside the thorax, leaving the stomach with its normal functions with regard to capacity and digestion.

8. The use of a colonic loop intrathoracically gives a better esthetic appearance to the individual and protects the loop.

9. Intrathoracic colonic esophagoplasty has the advantage of allowing any possible treatment on the diseased esophagus, extending the indications of this procedure to the treatment of some of the congenital lesions, and also to the malignant tumors of the organ.

SUMMARY

1. A case of generalized carcinoma of the esophagus presenting only a small mucous lesion at the level of the aortic arch is described.

2. A subtotal esophagectomy was performed without the possibility of the re-establishment of an esophagogastric continuity in one single operation.

3. Three months later an intrathoracic esophagoplasty was attempted by using a segment of the large bowel to re-establish the transit.

4. A careful review of the literature has not revealed a description of this intrathoracic technique in man.

5. This plastic operation may be considered feasible and more physiologic than esophagoplasties described up to the present time.

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THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

THIRTY-THIRD ANNUAL MEETING

MARCH 27, 28, AND 30, 1953. FAIRMONT HOTEL

SAN FRANCISCO, CALIFORNIA

PROGRAM

Friday Morning, March 27, 1953

9:00 A.M. BUSINESS MEETING

9:30 A.M. SCIENTIFIC SESSION

1. **Unilobar Obstructive Emphysema in Infancy Treated by Lobectomy.**
   Herbert Sloan, Ann Arbor, Mich.
2. **Mucocele, Congenital Bronchiectasis, and Bronchiogenic Cyst.**
   B. H. Ramsay, and Francis X. Byron, Los Angeles, Calif.
3. **Hydatid Disease as It Affects the Thoracic Surgeon.**
   M. P. Susman (by invitation), Sydney, Australia
4. **Surgical Considerations in Focalized Pulmonary Histoplasmosis.**
   James H. Forsee, and Thomas F. Puckett (by invitation), Denver, Colo.
5. **Extensive Bronchiectasis: A Clinical and Physiologic Evaluation of the Results of Surgical Treatment.**
   Edgar P. Mannix, Jr. (by invitation), Francis S. Gerbasi (by invitation), Charles E. O'Brien (by invitation), and Richard H. Adler (by invitation), Forest Hills, N. Y.
6. **Decisions in Thoracic Surgery as Influenced by Knowledge of Pulmonary Physiology.**
   Warriner Woodruff, Carl G. Merkel, and George W. Wright, Saranac Lake, N. Y.

Friday Afternoon, March 27, 1953

2:00 P.M. SCIENTIFIC SESSION

7. **Localization of Radioactivity in the Lung and Thoracic Lymph Nodes.**
   J. Ray Bryant (by invitation), and Harold F. Berg (by invitation), Louisville, Ky.
8. **The Ability of the Pulmonary Vascular System to Influence the Spread of Tumor Emboli.**
   Edwin A. Lawrence, Donald B. Moore (by invitation), and George L. Bernstein (by invitation), Indianapolis, Ind.
9. **Bronchial Resection and Anastomosis.**
   Paul W. Gebauer, Honolulu, T. H.
10. **Bronchiolar ("Alveolar Cell") Carcinoma of the Lung.**
    Clifford F. Storey, K. P. Knudtson (by invitation), and B. E. Lawrence (by invitation), St. Albans, N. Y.
11. **The Significance of Pulmonary Hypertension as a Cause of Death Following Pulmonary Resection.**
    W. E. Adams, John F. Perkins, Jr. (by invitation), and Adolfo Flores (by invitation), Chicago, Ill.

12. **Prevention of Chest Wall Defects: Use of Tantalum and Steel Mesh.**
Donald B. Effler, Cleveland, Ohio

Saturday Morning, March 28, 1953

9:00 A.M. SCIENTIFIC SESSION

13. **The Present Chaos Which Exists Regarding Pulmonary Resection.**
E. J. O'Brien, and Paul V. O'Rourke, Detroit, Mich.

14. **The Use of Isoniazid as a Prophylactic Antibacterial Agent in Pulmonary Resection for Tuberculosis: A Preliminary Report.**
Max E. Childress (by invitation), Mildred E. Thoren (by invitation), and Albert C. Daniels (by invitation), Weimar, Calif.

15. **The Influence of Long-Term Chemotherapy on the Surgery of Pulmonary Tuberculosis.**
John D. Steele, B. G. Narodick (by invitation), and A. V. Cadden (by invitation), Milwaukee, Wis.

16. **Segmental Resection of Pulmonary Tuberculosis: An Analysis of Three Hundred Cases Followed From One to Five Years.**
J. Maxwell Chamberlain, Robert Klopstock, and Charles F. Daniels (by invitation), New York, N. Y.

17. **Electrolyte Studies in Pericardial Resection.**
Walter B. Crandell, Andrew Yeomans (by invitation), David Hoffman (by invitation), and George H. Stueck, Jr. (by invitation), White River Junction, Vt.

18. **Bronchial Closure in Pulmonary Resection: A Clinical and Experimental Study Using a Pedicled Pericardial Fat Graft Reinforcement.**
Lyman A. Brewer, III, Frank S. Dolley, Edward L. King (by invitation), and Angel F. Bai (by invitation), Los Angeles, Calif.

Saturday Afternoon, March 28, 1953

2:00 P.M. EXECUTIVE SESSION

3:00 P.M. SCIENTIFIC SESSION

ADDRESS OF THE PRESIDENT—Robert M. Janes, Toronto, Ont.

19. **The Experimental Closure of Atrial Septal Defects.**
Harris B. Shumacker, Jr., and Thomas C. Moore (by invitation), Indianapolis, Ind.

20. **Surgical Treatment of Mitral Insufficiency: An Experimental Study.**
Max G. Carter, and John M. Gould (by invitation), New Haven, Conn.

21. **Pulmonary Valvuloplasty Under Direct Vision: The Aid of a Mechanical Right Heart.**
F. D. Dodrill, Robert A. Gerisch (by invitation), Aran S. Johnson (by invitation), and Edward Hill (by invitation), Detroit, Mich.

6:30 P.M. COCKTAILS AND DINNER—Fairmont Hotel.

Attendance limited to members of the Association and their wives, invited speakers and their wives. Dinner dress.

Monday Morning, March 30, 1953

9:00 A.M. SCIENTIFIC SESSION

22. **The Problem of the Hiatus Hernia of the Diaphragm.**
Conrad R. Lam, and Leo J. Kenney (by invitation), Detroit, Mich.

23. **Muscle Wall Tumors of the Esophagus.**
Richard H. Sweet, and Lamar Soutter, Boston, Mass.

24. **Reinforcement of Aortic Aneurysms by Wrapping: A Research Report.**
J. Karl Poppe, Portland, Ore.

25. **Clinical and Cytologic Changes in Patients Receiving Aerosol Trypsin and Desoxyribonuclease.**
Seymour M. Farber (by invitation), David A. Wood (by invitation), R. Daniel Gorman (by invitation), and Samuel Pharr (by invitation), San Francisco, Calif.

26. **Torsion Ballistocardiography in Cardiovascular Surgical Patients.**
C. Frederick Kittle (by invitation), Kurt R. Reissmann (by invitation), E. Grey Dimond (by invitation), and Paul W. Schafer, Kansas City, Kan.

27. **An Experimental Study of the Effect of Parasympathetic Denervation of the Lung on Pulmonary Artery Pressure.**
Victor H. Kaunitz (by invitation), and Murray N. Andersen (by invitation), Buffalo, N. Y.

Monday Afternoon, March 30, 1953

2:00 P.M. SCIENTIFIC SESSION

28. **The Role of the Bronchial Artery Circulation in the Etiology of Pulmonary and Pericardial Suppuration: An Experimental Study.**
Richard S. Hahn (by invitation), and Emile Holman, San Francisco, Calif.

29. **The Production and Repair of Interatrial Septal Defects Under Direct Vision With the Assistance of an Extracorporeal Pump Oxygenator Circuit.**
Bernard J. Miller (by invitation), Victor F. Greco (by invitation), Burgess A. Smith (by invitation), and John H. Gibbon, Jr., Philadelphia, Pa.

30. **The Oxygenator Pump in Total By-Pass of Heart and Lungs: Laboratory Evaluation and Clinical Use.**
James A. Helmsworth, Leland C. Clark, Jr. (by invitation), Sam Kaplan (by invitation), Roger T. Sherman (by invitation), and Thomas Largen (by invitation), Cincinnati, Ohio

31. **Experiences With Cardiopexy in the Treatment of Coronary Disease.**
Samuel A. Thompson, New York, N. Y.

32. **Cardiac Surgery Under Hypothermia.**
Charles P. Bailey, Brian Cookson (by invitation), William Likoff (by invitation), H. E. Bolton (by invitation), D. F. Downing (by invitation), and W. B. Neptune (by invitation), Philadelphia, Pa.

33. **The Management of the Dangerously Torn Auricle.**
William K. Swann (by invitation), Thomas L. Lomasney (by invitation), and Jorge Rodrigues-Arroyo (by invitation), Knoxville, Tenn.

PAPERS TO BE READ BY TITLE

1. **One-Stage Thoracoplasty Using the Trapp Adhesive Hemicast.**
Frederick L. Coddington (by invitation), Mt. Edgecumbe, Alaska

2. **A Comparison of Thoracoplasty and Resection Therapy in the Treatment of Pulmonary Tuberculosis.**
Thomas G. Baffes (by invitation), Phyllis Schlotterbeck (by invitation), and Paul T. De Camp, New Orleans, La.

3. **Replantation and Transplantation of Lobes of the Lung of Dogs.**
   Felix A. Hughes, Jr., John W. Polk (by invitation), Francis E. Foley (by invitation), and John R. Fox (by invitation), Memphis, Tenn.
4. **Clinical Evaluation of Cortisone, Tyral, and Intermittent Positive Pressure Breathing (IPPB) Therapy in Thoracic Surgery by Lung Function Studies.**
   George D. Boone (by invitation), Tucson, Ariz.
5. **Pectus Excavatum: Report of Five Additional Cases Treated Successfully With a Rib Strut.**
   Ralph A. Dorner, Des Moines, Ia.
6. **Esophagophrenic Syndrome.**
   Reuben I. Shapiro (by invitation), Detroit, Mich.
7. **Circulatory Status of Resected Tuberculous Pulmonary Lobes.**
   Raymond J. Barrett (by invitation), Mikio Masaki (by invitation), and J. C. Day, Detroit, Mich.
8. **Bronchovascular Variations in the Normal Lung.**
   W. E. Bloomer (by invitation), A. A. Liebow (by invitation), and M. R. Hales (by invitation), New Haven, Conn.
9. **Asymptomatic Lung Cancer.**
   John L. Pool, William L. Watson, and Henry D. Diamond (by invitation) New York, N. Y.
10. **Physiologic Observations Before and After Decortication of the Lung.**
    Walter G. Gobbel (by invitation), Allan Stranahan, and Ralph D. Alley (by invitation), Albany, N. Y.

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Original Communications

THE USE OF SUTURE CONSTRICTION OF THE UPPER LOBE IN THE TREATMENT OF PULMONARY TUBERCULOSIS (PAULINO PROCEDURE)

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THE ideal type of thoracoplasty for advanced pulmonary tuberculosis, while retaining the relative safety of the multistaged selective thoracoplasty of the Alexander type,[1, 2] should if possible increase its effectiveness and should, in addition, be a single nondeforming operation with a minimal loss of the costal cage, and a maximal preservation of respiratory function. It should be applicable if necessary to bilateral disease.

In the summer of 1949, it was the privilege of one of us (P. T. D.) to observe in Rio de Janeiro the performance, by Dr. Fernando Paulino of that city, of a modified partial thoracoplasty and pneumonolysis which seemed to approach this ideal. This operation had been developed by Dr. Paulino and performed by him in approximately 20 cases. A review of his cases[4, 5] convinced us that the procedure merited further trial. The procedure employs the oldest, soundest, and most accurate method of constriction in surgery, namely, the suture ligature.

Since Sept. 16, 1949, we have employed the procedure thirty-four times, in thirty-one cases with gratifying results. Performed as a single operation with limited resection of the costal cage, deformity has been slight, and the loss of respiratory function has been minimal. This procedure has permitted bilateral operations in 3 cases. There have been few serious complications and, although it is too early to reach final conclusions, there is evidence that the control of the disease is as effective as, and possibly more effective than, that following the more extensive rib resection of the Alexander type.

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Read (by title) at the Thirty-second Annual Meeting of The American Association for Thoracic Surgery at Dallas, Texas, May 8-10, 1952.

Collapse and compression of the diseased portions of the lung are obtained by a series of three No. 10 crochet cotton ligatures placed about the lung and the overlying parietal structures which have been freed from the chest wall according to the Carl Semb technique.[6] A limited rib resection is performed (1) to permit ready access for the pneumonolysis, (2) to obliterate the extrapleural space created by the pulmonary collapse, and (3) to prevent overexpansion of the lung below the level of the collapse.

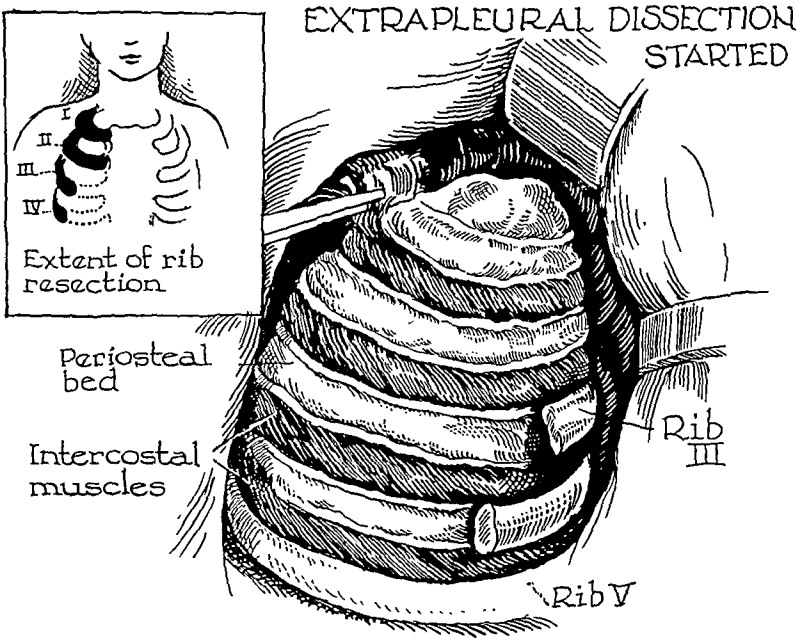


Fig. 1.—Technique of the Paulino procedure. Extent of the rib resection, and commencement of the extrafascial dissection, right lung.

TECHNIQUE

General anesthesia with intratracheal intubation is employed. We have placed the patient in the lateral decubitus position and employed a posterolateral parascapular incision. Dr. Paulino uses the face-down position.[4] Upon division of the muscles and retraction of the scapula, the rib cage is exposed. A subperiosteal resection of the first two ribs from the costal cartilages, the posterior two-thirds of the third rib, and the posterior half of the fourth rib is performed (Fig. 1). Posteriorly the ribs are removed only as far as the transverse processes of the vertebrae. The transverse processes are preserved. Beginning at the apex of the thorax an extrafascial pneumonolysis is performed by sharp and blunt dissection with division of the intercostal structures posteriorly and anteriorly (Fig. 1), according to the Semb technique.[6] The lung, overlying parietal pleura, and intercostal structures are freed to well below all gross lesions as determined by preoperative

roentgenography and operative palpation (Fig. 2). The dissection is limited inferiorly by the hilum of the lung, although it is possible to dissect somewhat below this point posteriorly if necessary to include lesions in the superior division of the lower lobe. If the collapse is carried to this point it may be wiser to remove a small posterior segment of the fifth rib.

The unique feature of the procedure is the method of achieving collapse, and preventing re-expansion, of the diseased lung. The apex of the pleura and the underlying lung is grasped with a large curved Kelly clamp. Beginning near the apex and concluding below the level of the grossly diseased lung, three concentric purse-string ligatures of No. 10 crochet cotton are placed about the lung and the overlying structures. Each ligature is caught in the pleura, the overlying tissues, or the lung itself at three or four points to prevent slipping. As each ligature is placed, it is drawn up snugly about the underlying diseased lung and tied (Fig. 3). Silver clips are placed on the lowest ligature as markers. At the conclusion of this stage of the operation all

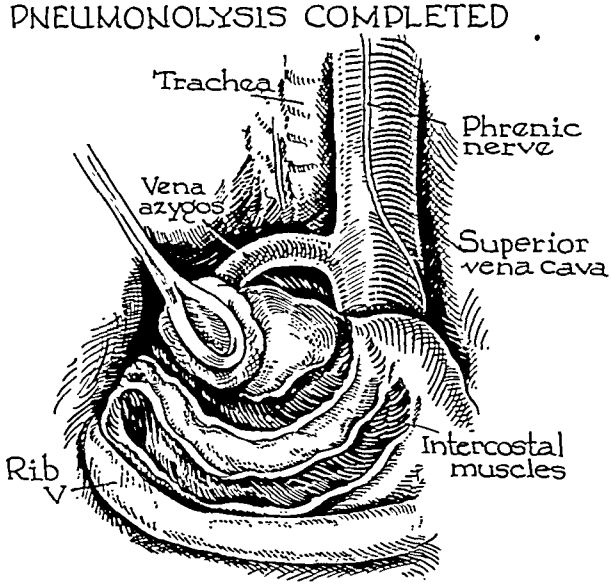


Fig. 2.—Technique of Paulino procedure. Pneumonolysis is completed to the hilum of the right lung.

of the grossly diseased portions of the lung have been uniformly and concentrically collapsed and compressed under direct vision in a far more accurate and dependable manner than is possible by rib resection alone. It should be emphasized that the degree of collapse of the lung is *not* dependent on the extent of the rib resection.

Laceration of the pleura does not unduly complicate the procedure. If the lung is not adherent to the parietal pleura care must be taken that the lung does not slip out as the ligatures are applied. A catheter may be introduced into the pleural cavity and either withdrawn at the close of the opera-

tion as the lung is expanded by the anesthetist, or it may be connected with a water trap for postoperative drainage. The wound is closed in layers with interrupted sutures of quilting cotton. A simple dressing is applied with a small pad placed in the anterior costal defect. The tendency toward paradoxical motion of the chest wall is slight.

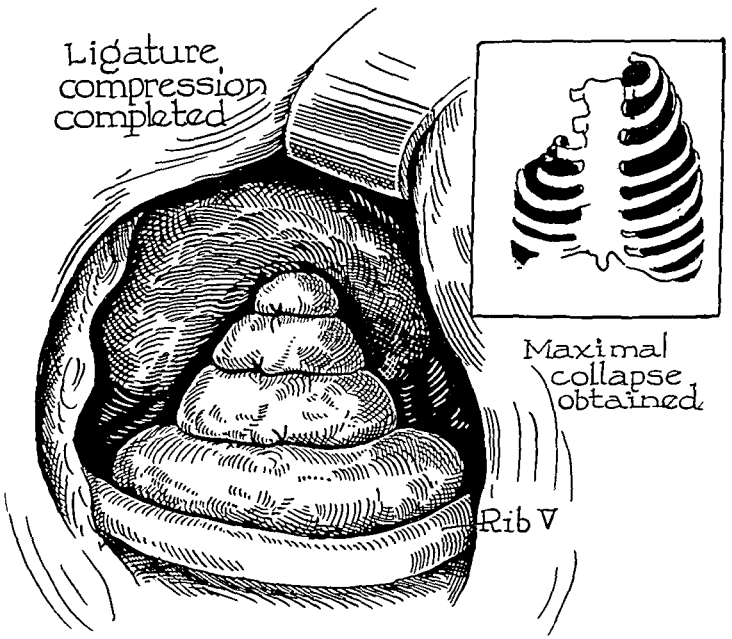


Fig. 3.—Technique of Paulino procedure. Ligature compression completed with demonstration of the maximal collapse obtained.

ADVANTAGES

The chief advantages of this procedure over the Alexander operation are: 1. Deformity is minimized. 2. There is maximal preservation of respiratory function. 3. It is performed in a single stage. 4. There is suggestive evidence that because of the more accurate application of collapse and compression better control of the tuberculosis is obtained. 5. It is applicable to bilateral disease.

INDICATIONS

The general indications for the operation are the same as for the standard type of thoracoplasty, and it may be elected in preference to the older procedure. Special indications for the Paulino procedure are: (1) the presence of bilateral advanced upper lobe disease, (2) the occurrence of unilateral upper lobe disease in a patient who has previously had contralateral thoracoplasty or lobectomy, (3) paramediastinal cavitary disease, (4) unilateral disease in the presence of limited respiratory reserve, and (5) a particular desire to avoid deformity as in a young individual.

CONTRAINDICATIONS

The general contraindications are the same as for the standard type of thoracoplasty. The special contraindications are: (1) the presence of advanced disease below the level of the hilum, (2) a large subpleural cavity, or (3) pleural disease. Ligature compression cannot be effectively applied below the level of the hilum but we have successfully controlled cavitary lesions in the dorsal division of the lower lobe. If absolutely necessary the lowest ligature may be placed on a bias to encompass posterior lesions lying slightly below the level of the hilum. Although we fear complications from ligature compression of a large subpleural cavity, the first patient upon whom we performed the procedure (J. M.) had a 6 cm. subpleural cavity which was successfully closed without untoward incident (Fig. 7, *A* and *B*), but we do not recommend the operation under these circumstances if alternative procedures are available.

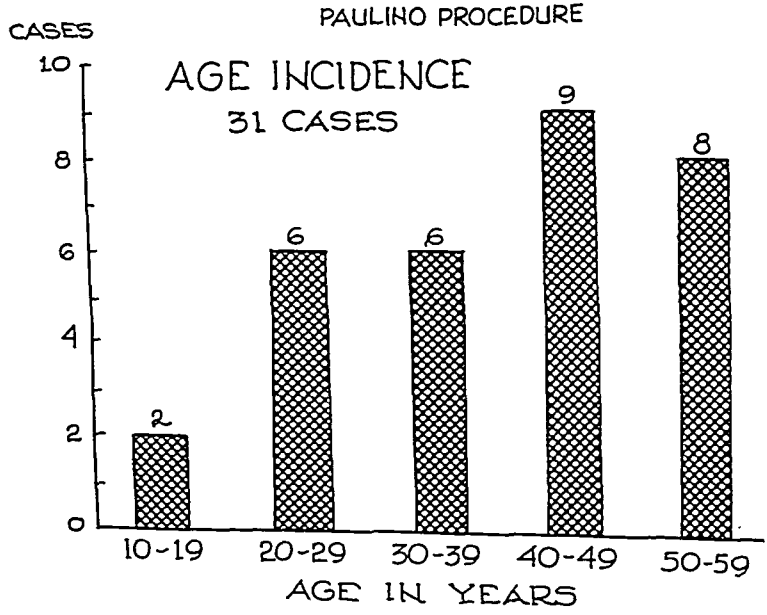


Fig. 4.—The age incidence by decades in thirty-one cases subjected to the Paulino procedure.

OPERATIVE EXPERIENCE

Thirty-four operations have been performed on thirty-one patients.* Of these twenty-eight were white, and three were nonwhite; twenty-three were men and eight were women. The age distribution of the cases is indicated in Fig. 4. Nine patients were in the fifth decade and 8 in the sixth, or a total of seventeen (55%) were more than 40 years of age. The three bilateral operations were performed in patients 28, 52, and 56 years of age.

*Twenty-three operations were performed at the Charity Hospital of Louisiana in New Orleans, seven at the Foundation Hospital, New Orleans, and one at the Mississippi State Sanatorium, Sanatorium, Miss.

The disease was unilateral in four cases, bilateral in twenty-seven, moderately advanced* in five, and far advanced* in twenty-six (Fig. 5). It is to be noted that as a group these cases represent advanced lesions for which either a major resection or an extensive thoracoplasty would have been required had this procedure not been used. On the operative side alone the disease was classified as moderately advanced in twenty-four and far advanced in ten.

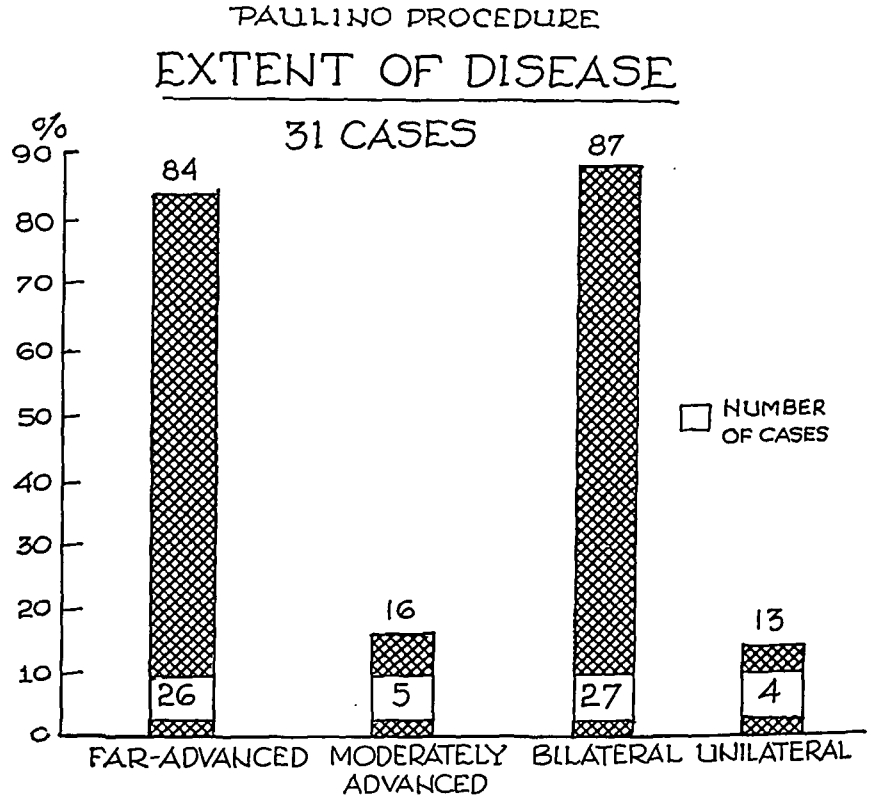


Fig. 5.—The extent of disease in thirty-one cases subjected to the Paulino procedure.

The Paulino procedure was chosen electively on general indications for thoracoplasty in thirteen cases, and on special indications for this technique in eighteen (Table I).

COMPLICATIONS

Serious complications of the operation have been infrequent (Table II). There has been no instance of bronchial fistula or of tuberculous infection of the pleural or extrapleural spaces. There has been no evidence of any of the ligatures cutting through, nor has there been any evidence of interference with the blood supply to the collapsed lung. The margin of safety between adequate collapse and interference with vascularity must be considerable be-

*National Tuberculosis Association Classification of 1950 is used throughout this report.

TABLE I. INDICATIONS FOR OPERATION IN THIRTY-ONE CASES

General indications	13 cases	
Special indications	18 cases	
Possible bilateral operation		7
Probable bilateral operation		6
Preservation of marginal function		6
Prevent deformity		1

cause the thirty-four operations have been performed by seven different operators, including five house officers. The pleura has been lacerated during dissection on eighteen occasions. This occurred more frequently early in the series when the extrapleural dissection was begun inferiorly rather than at the apex. The accident occurs most commonly through the very thin anterior pleura. Extrapleural fluid developed postoperatively in all cases. In eight patients the fluid had to be aspirated more than once and was considered excessive. Intrapleural fluid requiring aspiration developed in seven cases. Subcutaneous emphysema of moderate extent occurred twice. Nontuberculous infection of the extrapleural space developed twice, and of the intrapleural space once. In each instance it responded to simple open drainage.

TABLE II. POSTOPERATIVE COMPLICATIONS FOLLOWING THIRTY-FOUR OPERATIONS

COMPLICATION	CASES
Excess extrapleural fluid	8
Intrapleural fluid	7
Subcutaneous emphysema	2
Infection of extrapleural space	2
Empyema	1
Pulmonary embolism*	1
Respiratory failure*	1

*Fatality.

Two serious hemorrhages have occurred several months following operation. One patient (Case 10, M. L.) died of a sudden massive pulmonary hemorrhage five months after operation. Immediately after operation, a large contralateral paramediastinal cavity became apparent and remained open. The disease on the operative side appeared to be under control. The patient died at home and an autopsy was not performed. It was felt that the hemorrhage occurred in the large contralateral cavity. S. T. (Case 9) experienced severe pulmonary hemorrhage in April, 1951, five months after a left Paulino thoracoplasty and eight months after a similar operation on the right side. The disease was very extensive in both lungs, and it was felt that the collapse obtained at the first operation was technically not quite adequate (Fig. 8, *A* and *B*). This was the first operation performed by that operator. Although spread occurred in the right lower lobe following the hemorrhage, subsequently the disease has become inactive for eight months, and the patient has returned home (Fig. 8, *C*).

In Case 19 (F. H.) ipsilateral postoperative spread occurred. Bilateral apical cavitary disease was present and had not responded to prolonged bed rest with pneumoperitoneum. Either during operation or in the first few postoperative days, which were complicated by transient atelectasis and excessive

extrapleural fluid, the cavity slipped down below the lowest ligature. Subsequently a spread appeared in the ipsilateral lower lobe. This case constitutes a failure of the procedure although the patient has subsequently improved on further conservative treatment.

Two early postoperative deaths occurred. W. C. (Case 8) was ill-advisedly selected for operation as a heroic procedure in spite of very far-advanced bilateral disease extending below the hilum, and a limited respiratory reserve. Although his postoperative course was not otherwise complicated he died on the eighth postoperative day of respiratory failure. D. S. (Case 12) with far-advanced bilateral disease died of an apparent massive pulmonary embolism on the fourteenth day following the second of bilateral Paulino procedures. The disease had apparently been controlled on the first side and the sputum had converted, but a persistent cavity on the second side indicated the second operation. The one late postoperative death, from hemorrhage in the fifth month, has been referred to previously.

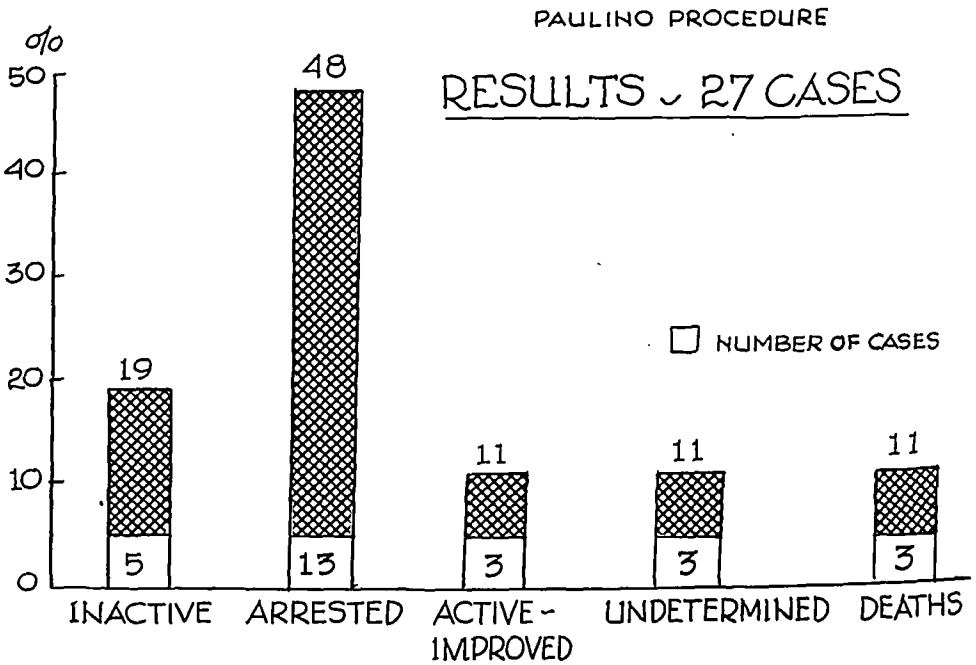


Fig. 6.—Results of the Paulino procedure in twenty-seven cases operated upon more than seven months ago.

RESULTS

Four of the thirty-four operations have been performed in the last four months. In each case adequate collapse has apparently been obtained and the patient is improved, but it is too early to evaluate the results. Two patients have been lost to follow-up. Of the remaining twenty-five patients, who were operated upon more than seven months ago, the disease has become *inactive* in five for periods of 6, 8, 18, 18, and 22 months (Fig. 6). The activity classifica-

tion is unlimited in three, class IV in one, and class III in one. The disease has been *arrested* in thirteen additional cases for 4.5 to 24 months with an average duration of arrest of 12.3 months. In nine of these cases sputum cultures are negative, and in only one case has there been a positive culture (one positive,

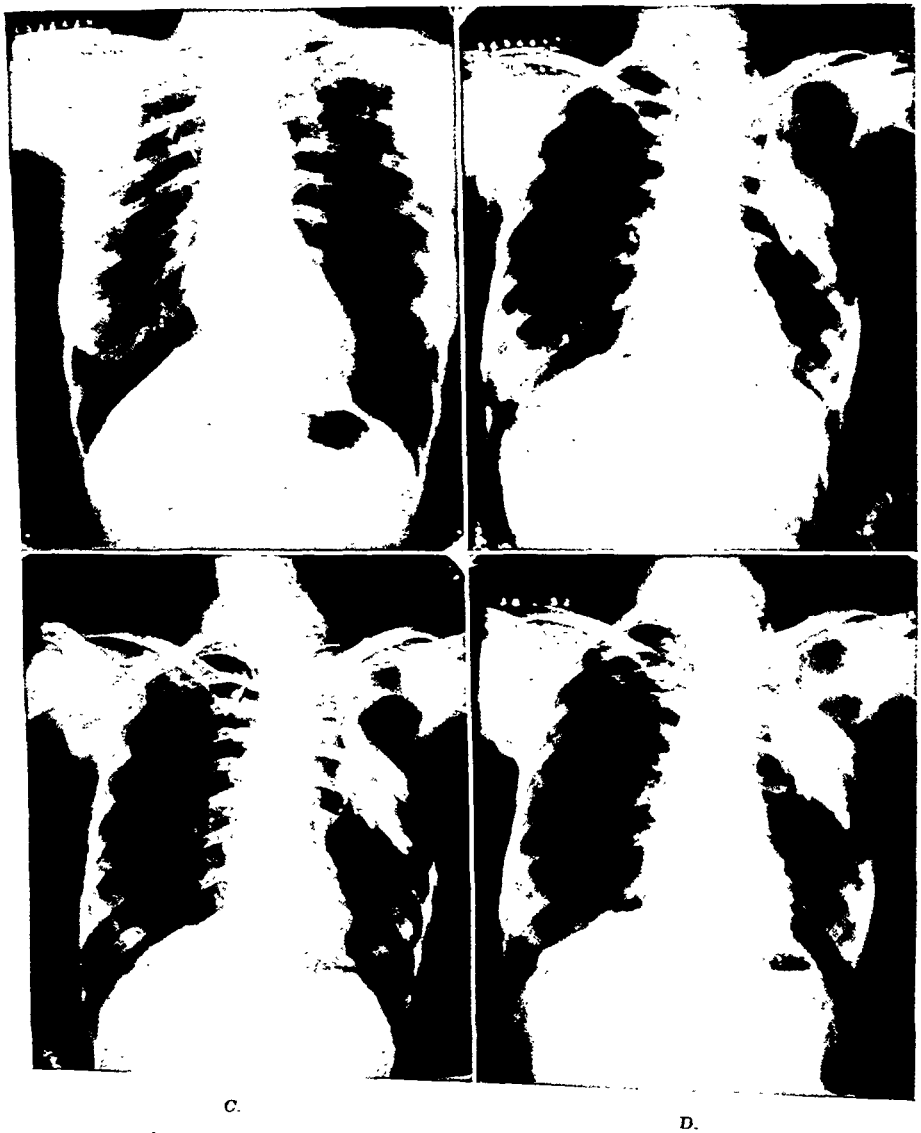


Fig. 7.—Case 1. J. M., a white woman, aged 36 years, with far-advanced bilateral tuberculosis. *A*, July, 1949, a large cavity in left upper lobe. Unsuccessful pneumothorax. Inactive contralateral disease. Left Paulino performed September, 1949. *B*, July, 1951, inactive status, 1 year. Moderate scoliosis. *C*, October, 1951, reactivation and cavitation in right upper lobe. Lesions on left side inactive. *D*, January, 1952, cavity closing on conservative treatment.

two negative) at the most recent checkup. The activity is unlimited in four, and class IV in nine cases. One patient (Case 26) who had negative sputum cultures and no clinical or x-ray evidence of activity on a single postoperative checkup four months after operation is classified, with the two patients who have not been followed, as activity undetermined (Fig. 6).

Three cases are classified as active, improved. The disease in Case 1 (J. M.) became inactive following an operation in September, 1949, and the patient returned to full activity. Two years later contralateral disease reactivated and a cavity formed (Fig. 7, *C*). This is responding to conservative treatment (Fig. 7, *D*). Roentgen studies, including planigraphy, fail to reveal activity on the operative side. F. H. (Case 19) is classified as active; the disease has improved on postoperative conservative therapy but, as previously described, this patient represents the one technical failure of the operation. F. B. (Case 27) had bilateral cavitary disease. Following operation in September, 1951, the disease has apparently been controlled on the operative side and the contralateral disease has improved so much that the originally contemplated bilateral operation may not be necessary. Recent sputum concentrates were negative.

The eighteen patients in whom arrested and inactive results were observed represent 67 per cent of the twenty-seven patients operated upon more than seven months ago, and 72 per cent of the twenty-five cases which have been followed. If Cases 1, 26, and 27, in which the results on the operative side appear to be entirely satisfactory, are added the operation has been beneficial to 78 per cent of the twenty-seven patients operated upon, and to 84 per cent of the twenty-five cases followed. Seven of these patients have returned to full activity. The two early postoperative deaths represent a case fatality rate of 6.4 per cent and an operative mortality rate of 5.9 per cent. With the third (late) death the over-all case fatality rate for the entire series of thirty-one cases is 9.7 per cent.

RESPIRATORY FUNCTION

An important advantage of this operation is the relative preservation of respiratory function. This was borne out by preoperative and postoperative total lung function studies carried out in twelve unselected cases (Table III). In ten cases following unilateral operation there was an average reduction in the vital capacity of 357 ml. In six cases following unilateral operation there was an average reduction in the maximum breathing capacity of 8.7 liters per minute. It is noteworthy that none of the three patients on whom bilateral operations were performed had significant dyspnea postoperatively. In the two in whom studies were made before operation and following the second operation (Table III) the maximum breathing capacity was reduced by 13 liters per minute in one, and 23 liters per minute in the second; and the vital capacity by 290 ml. in one, and 1,280 ml. in the other. Both of these patients have returned to normal activities without significant dyspnea.

ROENTGEN STUDIES

Careful roentgenologic studies in the follow-up period have failed to reveal the persistence of cavitation on the operated side in any case other than

TABLE III. PREOPERATIVE AND POSTOPERATIVE RESPIRATORY FUNCTION STUDIES

CASE	VITAL CAPACITY (ML.)			MAXIMUM BREATHING CAPACITY (LITERS/MINUTE)		
	PREOP.	POSTOP.	CHANGE	PREOP.	POSTOP.	CHANGE
			Unilateral Operation			
1	2,650	2,590	-60	--	90	--
2	1,985	1,550	-435	64	44	-20
3	1,490*	1,270*	-220	--	--	--
5	2,220	1,790	-430	60	49	-11
6	3,230	3,200	-30	99	88	-11
7	2,100	1,905	-195	--	72	--
11	4,060	3,420	-640	--	93	--
12a	3,000	2,460	-540	96	86	-10
22	1,640	1,410	-230	47	53	+6
27	3,070	2,280	-790	75	59	-16
	Mean		-357			-8.7
			Bilateral Operations			
9a	3,560	1,930	--	76	53	--
9b	--	2,280	-1,280	--	53	-23
15a, b	3,670	3,380	-290	104	91	-13

*Contralateral pneumothorax.

a—Indicates patient later had another operation.

b—Represents those patients in whom studies were made before the operation and following the second operation.

Case 19, previously mentioned, where the lung slipped out from under the ligatures. Studies have routinely included erect posteroanterior, lateral, and oblique views, Bucky films, and in nine instances, body section films. Significant scoliosis has developed in only one case (Fig. 7). In a few instances some confusion arose because the lung below the level of the collapse expanded upward, surrounding the collapsed tissues, and giving highlights suggesting cavitation (Fig. 9, *C* and *D*). The differentiation was readily made on study of lateral views and planigrams. Our first case, and two bilateral cases, are represented in Figs. 7, 8, 9.

DISCUSSION

It appears certain that until more effective conservative treatment is developed and all cases of tuberculosis are diagnosed and treated before destructive lesions have occurred, surgery will be called upon to assist in the control of the more advanced pulmonary lesions. Excision of such lesions is theoretically to be preferred to thoracoplasty, but in our experience excision has carried a higher mortality and a higher incidence of serious postoperative tuberculous complications.[3] Therefore we consider thoracoplasty indicated if it can be reasonably expected to control the lesions.

We have a great deal of respect for the multistaged, selective thoracoplasty of the Alexander type,[1, 2] which has proved to be effective, relatively safe, and largely free of serious postoperative complications. The procedure does have, however, several disadvantages. These are: 1. Multiple stages are required. 2. The procedure is frequently deforming. 3. A minimum of six ribs must be removed for adequate collapse. 4. To obtain satisfactory collapse of diseased portions of the lung it is necessary to remove two or more ribs below the level of the lesions thus causing considerable additional loss of pulmonary function. 5. The procedure can seldom be applied to bilateral dis-

ease or to contralateral lesions following lobectomy. 6. In spite of apparently adequate collapse, cavities in the operative area may remain open and the disease become active. It is debatable whether this persistent activity is due to technical inadequacy in the application of collapse and compression, or to inherent inadequacies in the principle of collapse therapy.



Fig. 8.—Case 9. S. T., a white man, aged 56 years, with far-advanced bilateral pulmonary tuberculosis. *A*, July, 1950, extensive cavitary disease in both upper lobes. Right Paulino procedure performed July, 1950. *B*, September, 1950, appearance following operation. Collapse may have been inadequate. Left Paulino procedure October, 1950. *C*, October, 1951, hemorrhage and right lower lobe spread in March, 1951. Subsequent response to antibacterial therapy. Disease inactive in April, 1952.

Numerous attempts have been made to modify, or to substitute for, this procedure in an effort to avoid these disadvantages. Partial thoracoplasty, extrapleural pneumonolysis with or without plombage, or a combination of the two procedures as in the Semb operation do indeed avoid many of these disadvantages. They have uniformly failed, however, in the most important

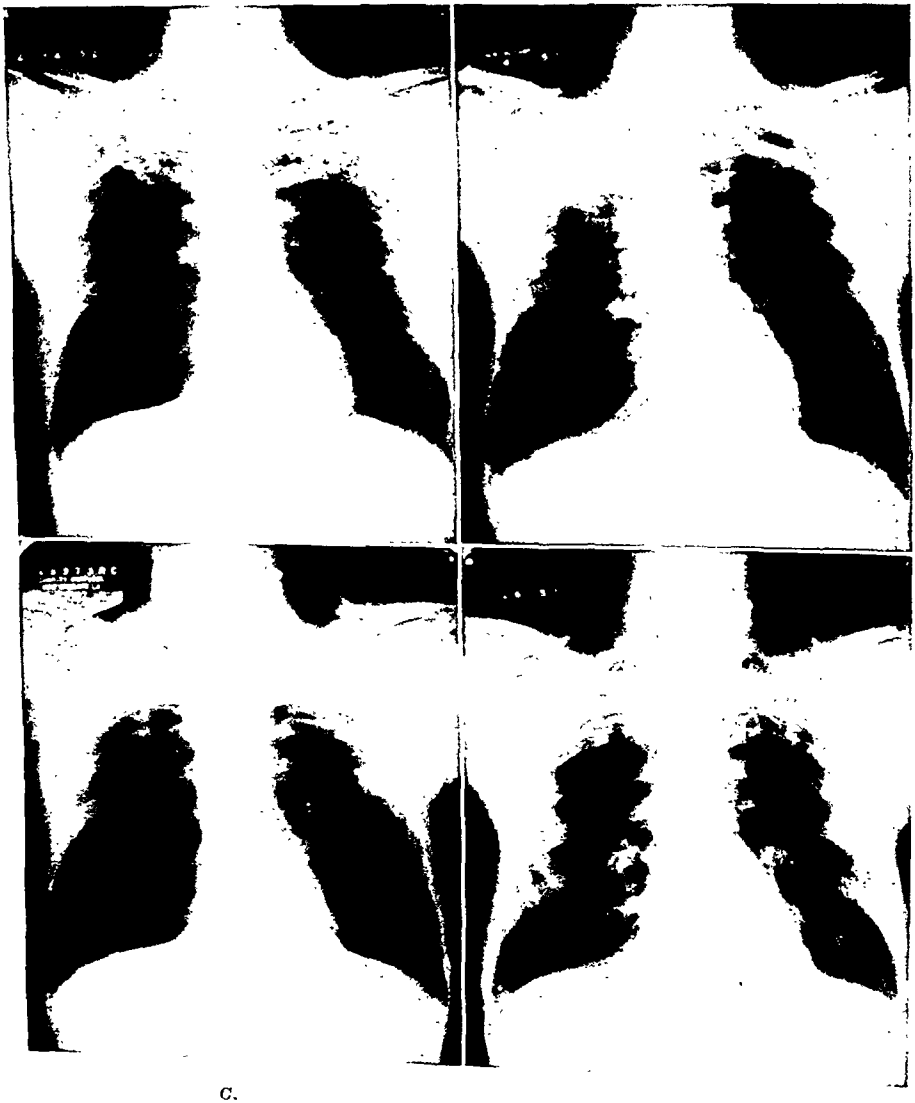


Fig. 9.—Case 15. L. B., a white man, aged 52 years, with far-advanced bilateral tuberculosis. *A*, December, 1950, extensive bilateral cavitary disease. Right Paulino procedure, December, 1950. *B*, April, 1951, good collapse of operative side. Persistent contralateral disease. Left Paulino procedure, April, 1951. *C*, May, 1951, good bilateral collapse. *D*, October, 1951, some overexpansion of lungs below the collapse causing highlighting which may be mistaken for cavities.

attribute of the Alexander operation and that is the control of advanced cavitary lesions in a high percentage of properly selected cases. We feel that these procedures fail either because of inadequate collapse or, even if the collapse is "adequate," because sole reliance is placed on the inherent ability of the diseased tissues to contract and to "control" themselves. The experience with intrapleural pneumothorax, extrapleural pneumonolysis, and partial thoracoplasty has amply demonstrated that if reliance is placed simply on the collapse of the diseased tissues onto the underlying elastic and mobile pulmonary tissue advanced destructive lesions of the lung will be controlled only sporadically. We feel that an important reason that the more extensive thoracoplasties are so much more effective is because they fix and compress the diseased tissues between the chest wall and the mediastinum, and the collapsed tissues are not affected by the activity of the lung below the level of the collapse. No one who has done a postthoracoplasty resection of the lung can doubt the factor of compression in an adequately performed thoracoplasty.

We tried this procedure chiefly because it avoided the pitfall of inadequacy common to all other conservative operations. The diseased tissues are collapsed, compressed, and fixed, and they are not affected by the activity of the uncollapsed portion of the lung. Our experience has indicated that it is a safe, single operation which minimizes deformity and loss of respiratory function, and which can be applied to bilateral pulmonary disease. It is too early to draw final conclusions concerning its ability to control advanced disease, but certainly the accurate application of concentric collapse and compression under direct vision should be as effective as the results obtained blindly by removal of the costal cage. Our experience to date indicates that the collapse is as effective as, if not more effective than, that obtained by the more radical operation.

The use of a ligature, so commonly employed as a crushing strangulating weapon, suggests certain horrifying complications when applied to a vascular structure such as the lung. These thoughts occurred to us, and we are sure that if the safety of the operation had not already been demonstrated by Dr. Paulino we would have been reluctant to try it. The margin of safety between adequate compression and dangerous strangulation must be great as the procedure was performed in our series by seven surgeons, five of whom were house officers. Serious troubles could arise if the ligatures were carelessly applied but it is necessary only to equal, or to slightly excell, the compression obtained by the standard thoracoplasty. Actually this operation does not introduce a new principle into the surgical treatment of pulmonary tuberculosis. It is a new and more accurate technical application of an old principle.

SUMMARY

A technique of partial thoracoplasty, extensive pneumonolysis, and collapse and compression of the lung by suture ligatures is presented. A thirty-one-month experience in thirty-one cases, including bilateral operation in three cases, indicates that this procedure is safe and reliable when applied to suitable

advanced tuberculous lesions of the upper lung. The operation is performed in one stage; costal resection, deformity, and loss of respiratory function are reduced to a minimum; and it has been applied successfully to bilateral disease.

The procedure is recommended particularly when bilateral surgical procedures seem indicated, or when the respiratory reserve is limited. As the operation has several additional advantages it may also be used by election rather than the more extensive multistaged selective thoracoplasty of the Alexander type.

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CLINICAL EXPERIENCE WITH THE WATER-SOLUBLE BRONCHOGRAPHY COMPOUNDS

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THE use of absorbable water-soluble contrast solutions for bronchography has attracted considerable attention in recent months. In fact, five different drug concerns have made products of this type commercially available for routine or investigational use. Sufficient individual clinical experience has not yet been gained to evaluate these compounds adequately. Consequently, it seems advisable to review the products available, to correlate some of the experience which has been gained, and to emphasize some serious complications which have resulted from their use.

The proprietary products now available do not vary significantly in their physiologic activity. They do vary, however, with regard to their chemical composition. All have as their contrast medium iodopyracet (the diethanolamine salt of 3,5-diiodo-4-pyridone-N-acetic acid) or a very closely related product. This material has for a number of years been marketed in this country as Diodrast.* It has been extensively used for intravenous urography and angiography, and its properties are well known. In 1948 Morales and Heiwinkle[1] described experimental and clinical studies with a combination of 35 per cent iodopyracet in 2.5 per cent carboxymethylcellulose (the sodium salt of cellulose ether). This material is now marketed by Astra under the trade name of Umbradil Viscous B. The product is a slightly viscous solution, about 500 centipoise (cps.), and does not have a consistency which would seem ideal for bronchography. However, very excellent bronchograms have been obtained with this material, as is attested by the recent reports of Atwell and Pedersen,[2] Filpse, Hedberg, and Krueger,[3] and others.[4]

Per-Abrodil "M," marketed by Bayer as Per-Abrodil "BR," is an identical product but with a viscosity adjusted to 3,500 to 4,000 cps. at 37° C. Reference will subsequently be made to the extensive work done in Germany with this substance.[5]

Two other available products, Ioduron B (Cilag)[6, 7] and Diodone Visquese (Guerbet),[8] have approximately the same concentration of iodine per cubic centimeter[3] (0.25 Gm.). The base in these compounds, glycolmethyl cellulose, results in a solution of much greater viscosity. Diodone (4,000 cps.) is rather jellylike in consistency and has a slight brownish discoloration. Ioduron (35,000 to 40,000 cps. in 2.5 per cent solution) is highly viscous, tends to precipitate crystals which redissolve on warming, and also has the brown color. We

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*Manufactured by Winthrop-Stearns, Inc.

have on various occasions attempted to identify free iodine as the cause for the discoloration in these two products but have been unable to do so.

The fifth product we have originally described as Methocel-Diodrast.[9, 10] It is now available for investigational use through Winthrop-Stearns, Inc. This material is composed of 50 per cent Diodrast in 1.75 per cent methylcellulose. It forms a clear, relatively viscous solution which compares in consistency with the iodized oils. Pure methylcellulose (4,000 cps.) was chosen for this particular product because of the extensive studies which have been carried out indicating that it is essentially nontoxic.[11-13] The majority of all our bronchograms performed to the present time have been made with contrast media compounded with pure methylcellulose. We have occasionally used a product prepared with carboxymethylcellulose but have felt that the degree of bronchospasm which accompanies its introduction is somewhat greater than with methylcellulose solution. It appears that the efficiency of carboxymethylcellulose, methylcellulose, and glycolmethylcellulose as thickening agents increases in this order at similar concentrations. We feel, therefore, that this irritative response is not due to the carboxymethylcellulose but rather to the lower viscosity of the carboxymethylcellulose solutions tried. This low viscosity allows for a more rapid absorption of the iodopyracet.

We have reviewed our first fifty-six bronchograms made with water-soluble media (Methocel-Diodrast in all instances) for the purpose of determining whether the technique, the contrast, and the bronchial filling were of such quality as to be consistent with a satisfactory bronchogram. Of these fifty-six cases, twelve were considered unsatisfactory. Seven resulted in a division of opinion, and thirty-seven were considered unquestionably satisfactory. Because of the division of opinion the seven questionable bronchograms were classified as unsatisfactory. This means that 66.7 per cent of the bronchograms reviewed were satisfactory and 33.3 per cent were unsatisfactory. Correspondence with others who have been using the water-soluble materials in this country has given us eighty-three added reports on their use. These reports indicated a marked division of opinion concerning the results obtained with these agents. Those who were enthusiastic with their use obtained satisfactory bronchograms, whereas those with a limited experience in their use obtained unsatisfactory bronchograms. In general, approximately one-third of the studies which were reported were considered unsatisfactory. This corresponds with our observations.

There are many factors which contribute to the success or failure of bronchography, even with such standard solutions as Lipiodol and Iodochloral. Therefore, it is essential that these results be compared with another series in which the iodized oils were used. One of us (A. J. N.) was, during military service, Chief of the Peroral Endoscopy Section in a large army hospital. During that time a record was kept of bronchograms performed and of the number which were unsatisfactory. A total of 528 bronchograms were made using Lipiodol, and of these 83 were unsatisfactory. This gives a percentage of 15.9 unsatisfactory bronchograms. It would seem, therefore, that it is possible to

obtain a satisfactory bronchogram more consistently with one of the nonabsorbable iodized oils. On the other hand, enthusiasm for the water-soluble media is somewhat greater among our European colleagues. Dr. Karl-Heinz Willmann reports on over 2,000 bronchograms with Per-Abrodil "M" 60 per cent Viscous, done for all kinds of pulmonary disease.[14] The large majority of these bronchograms were considered satisfactory and no untoward results were observed.

The explanation for the discrepancy between the results obtained with water-soluble and oil contrast media rests in a number of factors which are inherent in any of the water-soluble media presently available. First, the absorption of the contrast material, in all instances iodopyracet, is extremely rapid. Hence, to obtain results which are at all satisfactory it is necessary to become familiar with the use of these media. Second, the material is miscible with the bronchial secretions and, consequently, it tends to fill the peripheral bronchial tree confluently. This is in contrast to the picture with iodized oils in which the bronchial tree is outlined and "clubbing," due to blockage by retained secretions, is observed. (See Fig. 2 for this comparison.) One must, therefore, learn to read these bronchograms from a somewhat different point of view than is the case with the iodized oil. Third, and perhaps most significant, is the fact that these media tend to create a rather severe degree of bronchospasm.* This, of course, contributed considerably to the "unsatisfactory" group of films, as manifested by incomplete filling secondary to the explosive effect of coughing. Elimination of coughing is essential to satisfactory bronchography. This is a good deal more difficult to obtain with iodopyracet compounds as the bronchographic agent than with iodized oil. However, meticulous attention to the topical anesthetization of the tracheobronchial mucosa will improve the results when using a water-soluble agent. It is interesting that this response is not as marked in the individual with diseased bronchi as in the case of the individual with the more normal bronchial tree. We also feel that the bronchospasm is somewhat greater where the products utilize carboxymethylcellulose as a base because the viscosity is so low that the iodopyracet is more rapidly absorbed from the bronchioles and alveoli. This statement hypothesizes that the iodopyracet is the factor which leads to bronchospasm. The explanation is thought to be related to a reflex mechanism precipitated by rapid absorption, through a tremendous vascular bed, of a large amount of iodopyracet in a very short period of time. On several occasions we have seen a degree of irritation which exceeded mere coughing and resulted in a profound degree of bronchospasm and laryngospasm, even requiring intubation to maintain an airway. Suggestive evidence that iodopyracet is the stimulating factor is given by the observance of similar severe bronchospasm in a patient who died following the rapid intravascular injection of iodopyracet for angiocardiography.

The use of water-soluble media may also carry the danger of leading to a fatality. We have had two deaths associated with bronchography in which these materials were used. These cases should be reviewed at this time.

*Note: We have not proved that the cough, as clinical evidence of bronchial irritation, is secondary to bronchospasm but are assuming that this is true.

CASE REPORTS

CASE 1.—L. S., a white woman, 53 years of age, was admitted to Denver General Hospital on May 12, 1951, because of cough, weakness, and loss of weight. She also reported exertional dyspnea and small amounts of frothy yellow sputum. No allergic history was elicited.

On physical examination blood pressure was 110/60, pulse 120, and temperature 100.2° F. Auscultation of the chest revealed dry inspiratory and expiratory râles throughout both lung fields. Dullness was present in the right base posteriorly. The heart was enlarged to the left. Circulation time, arm to tongue, was 13 seconds, and the venous pressure was 70 mm. of water. Admission laboratory work was not remarkable aside from a white blood count of 11,400. X-ray examination of the chest showed a questionable enlargement of the right hilar area. Sputum was repeatedly negative for acid-fast bacilli. Bronchoscopy performed under topical cocaine anesthesia was negative.

On June 6, 1951, the patient was prepared for bronchography. She was given atropine 6 mg. on the ward and 50 mg. of Seconal Sodium intravenously upon arrival in the operating room. The pharynx and larynx were topically anesthetized with 20 c.c. of 5 per cent cocaine hydrochloride solution. A No. 14 French catheter was inserted into the trachea. Two cubic centimeters of 5 per cent cocaine solution was injected into both right and left main stem bronchi through the catheter. The patient was then taken to the x-ray department. Her condition appeared satisfactory. An intracutaneous test (0.1 c.c.) for sensitivity to Diodrast was negative. Fifteen cubic centimeters of 50 per cent Diodrast in 1.75 per cent methylcellulose was then instilled into the right bronchial tree. The patient was positioned and roentgenograms were taken. Fifteen cubic centimeters of the same material was then injected through the catheter into the left bronchial tree and the patient positioned. As the x-ray films were being taken the patient lost consciousness and had a convulsion. She was immediately returned to the operating room, where the pharynx was cleared of a small amount of the material and an endotracheal tube was inserted. At this time the patient had a second convulsion. Subsequently, respiratory motions ceased. She was given .5 Gm. of caffeine intravenously, and artificial respiration was maintained with oxygen under positive pressure from an anesthetic machine. Shortly afterward the heart beat became gradually weaker, and the patient was pronounced dead approximately thirty minutes after the initial convulsion.

The significant post-mortem findings were confined to the lungs, which both showed pulmonary edema, emphysema, and gross focal hyperemia. The report of the microscopic examination follows: The lungs showed focal areas of recent hemorrhage, in which the air spaces are packed with red blood cells. The septa were delicate, and the capillary spaces were filled with red blood cells, neutrophils, and eosinophils. The mucosa of the bronchi and bronchioles was intact. The submucosa was diffusely infiltrated with eosinophils and an occasional lymphocyte and mononuclear cell. In focal areas there was scattered a slight amount of anthracotic pigment. The kidneys revealed focal collections of eosinphils within the capillary spaces of the glomeruli. A Sudan fat stain of the adrenals revealed a normal component of fat. Sections of heart, liver, spleen, pancreas, adrenals, and brain revealed hyperemia but no other significant lesions.

Comment.—The pathologic summary* was as follows: "The finding of marked eosinophilia in the lungs was consistent with acute anaphylactic shock. Eosinophilic neutrophil infiltration of viscera has been described in acute adrenal insufficiency. However, this can probably be excluded by the demonstration of a normal fat component in the adrenals.

"Four volunteers, including myself, were used in an attempt to demonstrate a Prausnitz-Küstner reaction. The areas of skin passively sensitized with the patient's serum were tested with Demerol, cocaine, and the medium

*The post-mortem studies on this patient were conducted by Dr. Joseph R. Kraft, Chief Pathologist, Denver General Hospital.

used in bronchography. Negative results were obtained in each instance. Since passive transfer of drug sensitivity is rarely demonstrable, a negative P-K reaction does not exclude it. Toxicologic examination of vital viscera for cocaine demonstrated only minute amounts, so death due to overdosage, but not to sensitivity, can be excluded.

"Therefore, this is a case of a 53-year-old white woman who came to sudden death during a bronchographic procedure. The autopsy findings exclude to my satisfaction all causes of sudden death except that due to anaphylactic shock. Of the drugs given to the patient, none can be explicitly implicated nor excluded."

Certain clinical factors may also be of some significance in the final evaluation of this patient. She had previously received cocaine topically for bronchoscopy without any evidence of sensitivity, although on the occasion of bronchography she did receive 1.2 Gm. of the drug. Moreover, the time relationship between the last administration of cocaine and the initial convulsion (approximately fifteen minutes) would hardly be consistent with a cocaine reaction. On the other hand, the time relationship between the injection of Methocel-Diodrast and the convulsion (approximately five minutes) is consistent with a reaction to one of the components of this solution. Previous intracutaneous testing for sensitivity to Diodrast, while not conclusive, would tend to free this drug from implication. The evidence at hand, therefore, is suggestive enough to lend suspicion to methylcellulose as the causative factor in the anaphylactic response.

CASE 2.—B. S., a white woman, aged 69 years, was admitted to Colorado General Hospital, Tuberculosis Service, on Oct. 10, 1951, because of a chest survey film which was reported as showing far-advanced pulmonary tuberculosis. She denied all symptomatology and had had no weight loss. Past history revealed that the patient's first husband died of tuberculosis during World War I. The patient then remarried and had lived with her second husband twenty years, during which time she considered herself completely asymptomatic. The family, however, suspected tuberculosis because the patient had a constant chronic cough. No allergic history was elicited.

Physical examination revealed a well-developed individual in no distress. The trachea was deviated to the right. There was dullness to percussion over the medial portion of both lung fields posteriorly. Sputum and gastric washings were examined over a four-month period and were consistently negative for acid-fast bacilli. X-ray examination showed a contracted right upper lobe, within which were areas of radiolucency thought to represent cavitation. The mediastinal structures were shifted to the right. Several radiolucent densities, thought also to be cavities, were apparent in the left mid-lung zone near the hilus. Tomograms subsequently confirmed these findings.

First strength P.P.D. was negative. Second strength P.P.D. was faintly positive. Skin tests for histoplasmosis and coccidioidomycosis were negative. Three bronchoscopies were performed with consistently negative results except for the rather marked deviation of the trachea to the right.

The patient's course on the ward was uneventful. She remained afebrile and without complaint. During four months of observation she gained sixteen pounds.

On Feb. 5, 1952 bronchography was performed. The pharynx and larynx were topically anesthetized with 5 per cent cocaine. A No. 16 catheter was introduced through the larynx into the right main stem bronchus. One cubic centimeter of 5 per cent cocaine (total amount of cocaine used was less than 1 Gm.) was introduced through the catheter

for further anesthesia. Fifteen cubic centimeters of 50 per cent Urokon Sodium in 1.75 per cent methylcellulose was then injected into the right main bronchus. The patient did not cough and had no complaints. About ten minutes after the introduction of the contrast medium, and while waiting for the original films to be developed, the patient stated that she wanted to lie down. The patient's color was noted to be bluish-gray, and the pulse was rapid and thready. Almost immediately she became unresponsive and the pulse imperceptible. Subsequently respiration ceased and a frothy foam appeared from her mouth. The mouth and pharynx were aspirated, and intracardiac stimulants were given but were unsuccessful in the face of the marked and rapid circulatory collapse.

Post-mortem examination revealed the trachea and both main bronchi to be filled with bloody mucus. The mucosa of the trachea and right main bronchus showed hyperemia and petechial hemorrhages over an area corresponding to the distribution of the dye on the bronchogram (Fig. 1). The smaller bronchi on the left contained a clear, viscid, mucoid material. The upper lobe of the right lung appeared atelectatic and the bronchi compressed. The middle and lower lobes were red, heavy, and wet. Large portions of the lung parenchyma were replaced by irregular areas of firm gray tissue resistant to cutting.

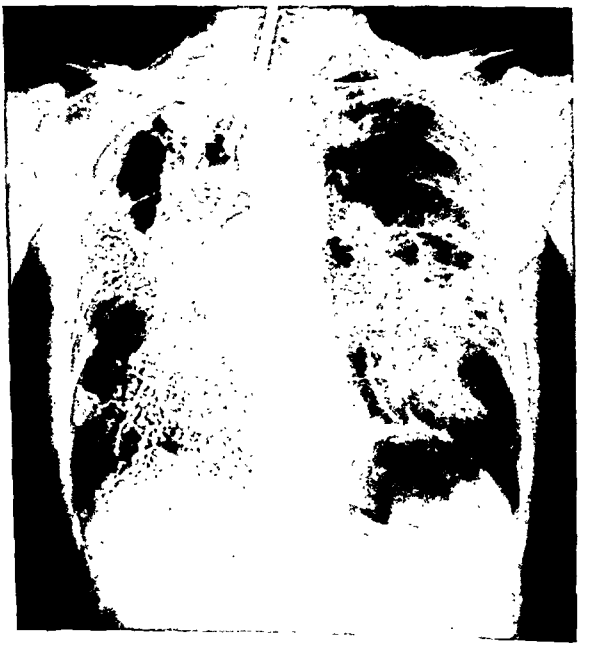


Fig. 1.—Bronchogram of the patient (Case 2) made prior to death in circulatory collapse. The distribution of the medium within the tracheobronchial tree corresponded to the area of mucosal hyperemia and petechial hemorrhage observed at post-mortem examination.

The cavities described by x-ray films had irregular necrotic walls and were filled with purulent exudate. The bronchi of the right lung were edematous and contained petechial hemorrhages in their walls and gross hemorrhage in their lumens. The surrounding lung was very hyperemic and edematous, with areas of frank fresh hemorrhage into the alveoli.

Microscopic examination revealed the cavities to be large bronchiectatic cavities with secondary abscess formation. The remains of the bronchiolar wall could be identified around the abscess. The cavities were filled with segmented neutrophils, and the walls were densely infiltrated with neutrophils and phagocytes. None of the cavities had the appearance of caseation necrosis.

The lesions in the lungs were granulomatous, composed of giant cells, epithelioid cells, a few lymphocytes, macrophages, and areas of fibrosis. The giant cells were atypical, many having central nuclei and containing inclusion bodies. Gömöri stains showed the areas which had been interpreted as necrosis to be actually areas of fibrosis. Intact collagen fibers extended through the lesions. The reticulum stains showed reticulum fibers arranged in whorls around the central giant cells in many of the lesions and merging with the collagen fibers in the fibrotic areas. Tubercle-like lesions, consisting of giant cells, and stellate-arranged epithelioid cells were found in lymph nodes from the neck, chest, and abdomen, in the spleen, and in the myocardium. Circumscribed areas of fibrosis were present in the liver.

The interpretation of these findings led our pathologists to the following conclusions: The cavities seen in the x-ray pictures were actually large bronchiectatic cavities with secondary abscess formation. The granulomatous lesions, when considered in conjunction with the history, laboratory findings, anatomical location, and specific characteristics, were those of sarcoidosis rather than tuberculosis. The clinical picture, the time factor, and the post-mortem findings support the diagnosis of death due to an anaphylactoid reaction to the substance used for bronchographic study.

Comment.—The nature of this patient's death leaves little doubt but that it was caused by an anaphylactoid type of reaction. The distribution of the hemorrhagic reaction within the wall of the tracheobronchial tree, when compared with the bronchogram, certainly implicates the medium which was used for bronchography. Unfortunately, the patient was not previously tested for sensitivity to the Urokon or the methylcellulose. However, the literature now available does not reveal any reports of serious reactions to Urokon. It is being extensively used for urography, and we have been unable to obtain information of any serious unreported reactions. We have been using it in 70 per cent concentrations for aortography and have found it to cause essentially no reactions, vasospasm, or pain, even under local anesthesia. Moreover, experience has shown it to be so rapidly absorbed from the lung that the symptoms of sensitivity to this drug should have appeared much sooner if it were to be related to the reaction observed in this patient. On the other hand, methylcellulose would be expected to be absorbed more slowly. It is primarily disposed of by the lymphoid elements and the reticuloendothelial system. This system was primarily involved, as is evidenced by the areas of hyperemia and actual hemorrhage within the spleen. A similar hyperemia was observed in the liver, and the liver sinusoids were dilated. It seems reasonable to assume, therefore, that death in this instance could be related to the use of the methylcellulose, even though we have no direct evidence in this regard.

DISCUSSION

The concept of a water-soluble agent which will disappear from view shortly after the x-ray examination is especially appealing for tuberculous patients[15] as well as in routine bronchography.[16] Residual iodized oil remaining in the lung is a definite handicap to future study of the pulmonary tree. We feel that the water-soluble agents now available are helpful in overcoming this difficulty (Fig. 2). Nevertheless, they cannot be used without an awareness of the complications and difficulties related to their use. These difficulties might be grouped into three categories: namely, technical, toxic, and latent tissue reactions.

The technical difficulties are primarily associated with the rapidity with which the contrast medium is absorbed. The medium is taken up from the pulmonary tree with such great rapidity that within a few minutes practically none of the contrast substance is visible. Therefore, teamwork is essential between the physician performing the bronchography and the x-ray technicians. It is also almost impossible to fluoroscope these patients during the procedure of bronchography because of this inherent characteristic.

Bronchograms made with these media do not result in as high a degree of contrast as do iodized oils. This may be advantageous to a certain degree, since small parenchymal lesions will not be obscured by the medium. We have, however, made numerous attempts to improve this factor somewhat.



Fig. 2.—The x-ray films demonstrate, on the same patient, the comparison between iodized oils (Lipiodol) and water-soluble medium (Methocel-Diodrast) in bronchography. The film on the left shows a bronchogram of the right chest made with Lipiodol Jan. 12, 1951. On Feb. 27, 1951, a bronchogram of the left chest was made with absorbable contrast medium. The two films are comparable in their demonstration of the bronchial tree, although the Lipiodol gives a more striking contrast. However, the retained oil remains to confuse future evaluation on roentgenogram of the right lung, whereas the left lung was completely clear of the contrast material within a very short time.

Essentially, every water-soluble, radiopaque substance known has been studied.[17] Tetraiodophenolphthalein might seem to be ideal, since it is a compound containing four iodine atoms to the molecule in contrast to the two atoms of iodine in iodopyracet. However, this particular material is not miscible with the high molecular carbohydrate, methylcellulose, which we have been using as a base. This is apparently due to the very considerable difference in pH of the two substances, and it is necessary to choose a substance with a pH approximating that of the body to prevent damage to the tracheobronchial mucosa. A similar situation exists with sodium iodide as it relates to miscibility. Of the barium salts only barium sulfate is nontoxic, but because of its physical properties it is not suitable. Thorotrast might seem to be an ideal substance except that it is not absorbed from the pulmonary parenchyma as are other water-soluble media.[18] Instead, it apparently remains in

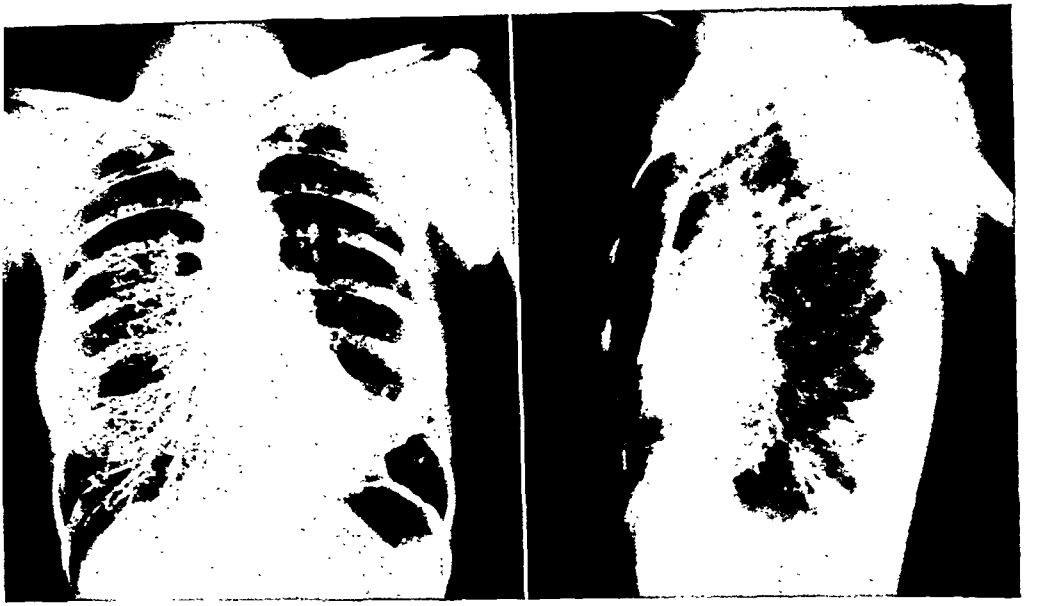
the reticuloendothelial system of the lung. This retention in the face of its mild radioactivity is a disadvantage. Of the remaining substances, Neo-Iopax and Skiodan seem to have no particular advantage over Diodrast. Lately we have been very interested in Urokon Sodium* because of its reportedly low toxic manifestations and because Urokon Sodium contains 65.8 per cent iodine per molecule as compared with 49.8 per cent iodine per molecule in Diodrast.[19-22] Initial bronchograms in human patients using 35 per cent Urokon Sodium in 1.75 per cent methylcellulose have given us the impression that there is a slight improvement in contrast with this particular medium (Figs. 3 and 4). Further studies are being carried out with this substance at the present time. However, it cannot be anticipated as being the final answer because it is absorbed at about the same rate as is iodopyracet.

The toxic manifestations of the water-soluble media add a certain degree of hazard to their use. As we have pointed out earlier, the water-soluble compounds seem to create a marked degree of bronchospasm, particularly in those individuals who do not have a severely diseased bronchial tree. Moreover, it may well be, in certain individuals, that there is a localized allergic response to the high molecular carbohydrate base or to the contrast substance utilized. Evidence is suggestive in this regard but not conclusive.

The severe degree of bronchospasm can often be overcome with adequate anesthetization. It has been our practice to inject about 1 c.c. of 5 per cent cocaine directly into the main stem bronchus of the side to be examined, just prior to the introduction of the bronchographic medium. This has considerably alleviated the degree of bronchospasm elicited in certain individuals. Unfortunately, it does not always prove effective. This method of introducing cocaine directly into the bronchial tree may in itself result in certain hazards. Experimentally it is known that the rapid assimilation of low concentrations of cocaine may be more toxic than slow assimilation of high concentrations of the drug. Death can, therefore, ensue from endobronchial anesthetization in this manner. It is our impression at the present time that Urokon Sodium does not precipitate the bronchospasm observed with the use of iodopyracet. At least, in no instance have we observed any unusual stimulation of the cough reflex when Methocel-Urokon has been used.

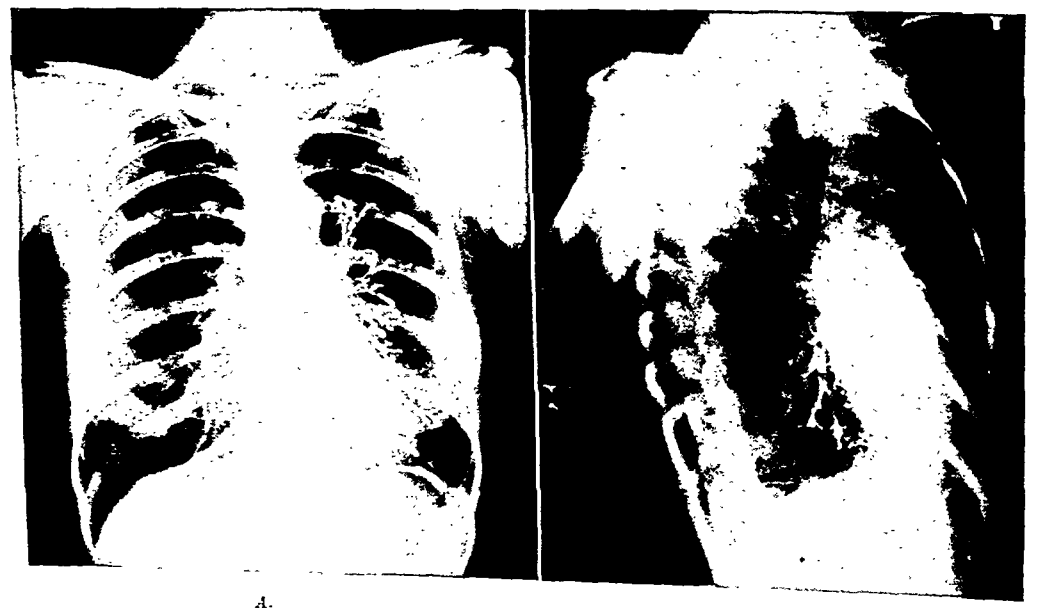
The latent effects of the water-soluble agents upon pulmonary tissue are not clearly understood. Vischer has reported the occurrence of a fibrotic reaction surrounding small foci of carboxymethylcellulose and glycolmethylcellulose which were retained in the pulmonary parenchyma.[23] Experimentally in dogs we were unable to detect any early evidence of an inflammatory reaction or any early evidence for retention of methylcellulose within the pulmonary tissues.[9] However, it is altogether conceivable that these high molecular compounds are retained to some degree within the pulmonary tissue. They are known to be engulfed by the reticuloendothelial cells, particularly in the liver and spleen, when introduced intravenously in large amounts.[11, 12] There is no reason to suppose that they might not be similarly engulfed in the reticu-

*Urokon Sodium is the trade name for sodium 3-acetylamino-2,4,6-triiodobenzoate; it is produced by the Mallinckrodt Chemical Works.



A. *B.*

Fig. 3.—*A* and *B*, Bronchograms illustrating the results which can be obtained with 35 per cent Urokon Sodium in 1.75 per cent methylcellulose. We feel that the contrast obtained on x-ray pictures with this material is superior to other water-soluble media and that the complicating bronchospasm is considerably less. (The films demonstrate clearly bronchiectasis limited to the segments of the left lower lobe and lingula.)



A. *B.*

Fig. 4.—*A* and *B*, See legend for Fig. 3.

loendothelial system of the lung following endobronchial introduction. In fact, Helmström and Holmgren did observe such a response with the endobronchial introduction of carboxymethylcellulose in rats and rabbits.[24] It is improbable, though, that all of this high molecular compound is absorbed. We feel that a great deal of this material is coughed up and expectorated, as is true with the iodized oils.[25] That which remains may act as any substance which is introduced into the bronchopulmonary tree and which is retained; that is, it is likely to give a latent reticular or fibrotic reaction around the retained nidus. This is true after the introduction of Lipiodol,[26, 27] yet it does not seem to be a contraindication to the use of this material. It does not seem to us, therefore, that this fact can be considered a serious deterrent to the use of the water-soluble compounds.

It can justly be said that no compound yet available has all the ideal characteristics for a bronchographic medium. The current water-soluble compounds are a step in the right direction. They must be used with caution and understanding as to their intrinsic dangers and limitations. They are an adjunct in our armamentarium for the diagnosis of pulmonary disease. They are not, however, the final answer and should not preclude further extensive study to find the more ideal agent for use in bronchography.

SUMMARY

The current status of water-soluble absorbable media for bronchography has been reviewed.

The advantages and disadvantages of these media have been enumerated and discussed.

A review of fifty-six consecutive bronchograms performed with Methocel-Diodrast reveals that this medium has been satisfactory in 66.7 per cent of the cases in which it was used and unsatisfactory in 33.3 per cent of the cases. These results have compared favorably with the information gained from correspondence among others in this country who have been interested in the use of the water-soluble compounds. We have also compared these results with 528 bronchograms performed with Lipiodol and find that in general iodized oils will give satisfactory bronchograms more consistently. Of these 528 cases, only 15.9 per cent were considered unsatisfactory.

Two deaths are reported which followed bronchographies in which Methocel-Diodrast and Methocel-Urokon were used. It is our opinion that the high molecular carbohydrate base precipitated the fatal anaphylactic reactions.

The inherent difficulties with the water-soluble media seem to be factors which are related to technical, toxic, and latent pulmonary changes associated with the use of these drugs.

The currently available water-soluble compounds for bronchography, when used intelligently, are a valuable adjunct to the diagnosis of pulmonary disease. They cannot, however, be considered the ideal agent for bronchography.

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THE CLINICAL SIGNIFICANCE OF CAVERNOLITHIASIS

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REPORTS dealing with pneumoliths or pulmonary calculi are concerned primarily with the broncholith. Concretions of this type are usually produced by intrabronchial perforation of caseous and partially calcified lymph nodes of the bronchopulmonary or tracheobronchial lymphatics. Schmidt and his associates[3] and Head[1] have recently reviewed their experience and that of others with broncholithiasis and have stressed its clinical significance. Another specific type of pneumolith is located within the lumen of pulmonary cavities and may therefore be termed cavernolith. The purpose of this presentation is to call attention to the possible clinical significance of such intracavitary concretions in the course of pulmonary tuberculosis.

From an etiological standpoint, cavernoliths probably have an origin similar to broncholiths, that is, from a partially calcified tuberculous focus.[1, 3] Calcifications of peripheral caseous foci in the lungs are a frequent occurrence and are most likely to occur in childhood or adolescence but may also occur during the course of adult tuberculosis.[2] In the event the caseous material in such a partially calcified focus discharges into the communicating bronchus, the lesion excavates, and the larger fragments of calcium remain as calculi which lie inside of the resulting cavity. Calcification of cellular detritus and other debris within a cavity offers a possible, but not likely alternate etiological origin for cavernoliths.

Although one would expect cavernolithiasis to be encountered frequently at autopsy, the significance of this condition in the course of pulmonary tuberculosis must have escaped attention as evidenced by the lack of clinical reports in the literature dealing with the subject. For this reason the following brief case reports* are submitted in order to illustrate some of the more important features in regard to the clinical course, diagnosis, and therapy in patients with pulmonary tuberculosis complicated by cavernolithiasis.

CASE REPORTS

CASE 1.—L. M., a 37-year-old man, was symptomless at the time that bilateral upper lobe cavities were discovered in May, 1950. Sputum examination was strongly positive for mycobacterium tuberculosis. Serial roentgenograms (Fig. 1, *A* and *B*) revealed the presence of a flaky cavernolith in the right upper lobe cavity. After a period of six months with conservative management, surgical treatment was advised. Curiously enough, the roentgenograms and laminograms made of the chest prior to surgery did not demonstrate the cavernolith and it was presumed that the fragment had been coughed up. Because of this, first stage thoracoplasty with apicolysis was performed on Oct. 6, 1950. The postoperative course was stormy. There was fever, massive hemoptysis with a marked "bronchial spread" to the middle and lower lobes on the right, and scattered dissemination to the left lung (Fig. 2).

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A. *B.*

Fig. 1.—Roentgenograms of the chest (Case 1) demonstrating a cavity with overlying pleural thickening in the right upper lobe. In *A*, the calcified fragment in the cavity is almost horizontal and in *B*, made two months later, the cavernolith is upright.

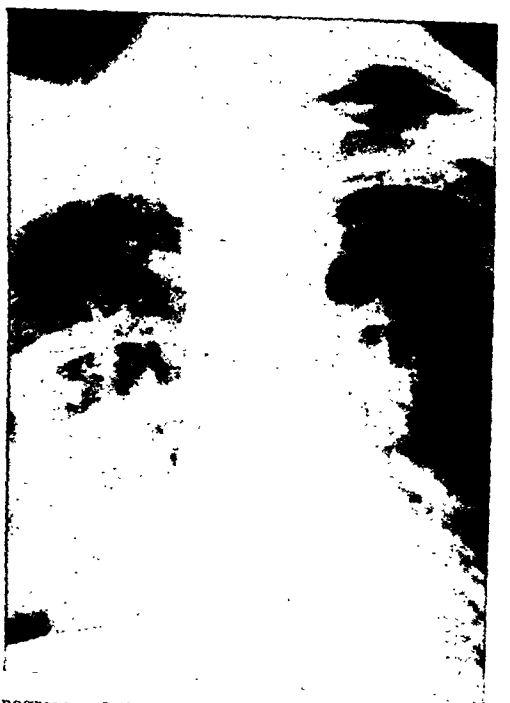


Fig. 2.—Roentgenograms of the chest (Case 1) made two months after first stage right upper thoracoplasty with apicolysis. There is marked contamination of the lower lobes bilaterally as a result of the severe postoperative hematemesis. The cavernolith had changed position again in comparison to Fig. 1.

Postoperatively, the cavernolith was well demonstrated in the right upper lobe cavity (Fig. 2). The patient recovered from this severe setback after a prolonged course of streptomycin and para-aminosalicylic acid. On Oct. 15, 1951, a right upper lobectomy was performed* and the patient recovered uneventfully. In the resected specimen (Fig. 3) the razor-sharp cavernolith was lying free within an unhealed cavity which measured 2.5 by 2 by 1.5 cm. in diameter.



Fig. 3.—Photograph of sectioned specimen of right upper lobe (Case 1) showing the cavity and the razor-sharp cavernolith which had been repeatedly demonstrated by roentgenography.

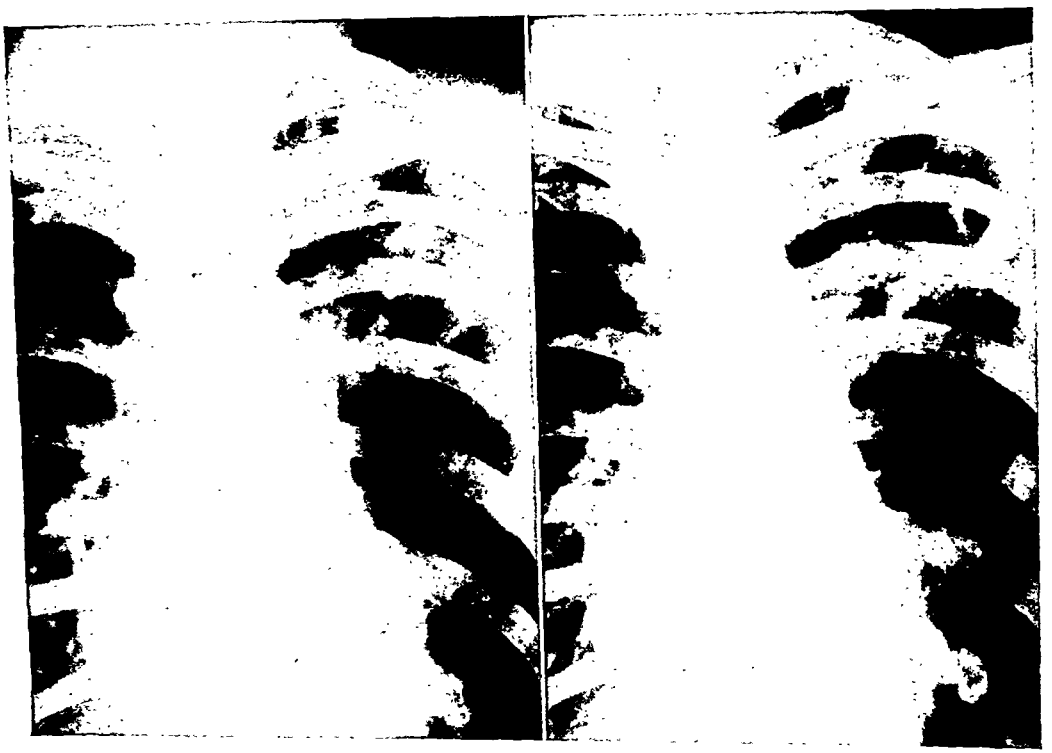
CASE 2.—R. H., a 41-year-old man, was seized suddenly with fever, malaise, and hemoptysis in September, 1945. Roentgenograms revealed cavitation with calcification in the left upper lobe. In the course of the next six months there were repeated similar episodes, and on one occasion he expectorated a tiny fragment of calcified material. During an attack, symptoms could be relieved only by strict and absolute bed rest. Roentgenograms which have since been destroyed indicated that the size and shape of the cavity varied continuously and that the relative position of the cavernoliths changed.

After a long and complicated course, both in and out of the hospital, thoracotomy was performed on July 17, 1946. At this time a left pneumonectomy was done† and the patient recovered uneventfully. The specimen contained multiple cavernoliths, one of which was loosely impacted in a small bronchial opening at the base of the cavity in the left upper lobe.

CASE 3.—R. L., a 34-year-old man, a tutor, was discovered in 1942 to have pulmonary tuberculosis with cavitation in the posterior segment of the left upper lobe. On restricted

*By Mr. Tubbs.
†By Mr. Brock.

activity he did well, but after an episode of strenuous exercise in May, 1949, he became ill again for a short period. During the next twelve months roentgenograms revealed that the left upper lobe cavity periodically filled with secretions and subsequently discharged into the bronchus. The sputum was intermittently positive for mycobacterium tuberculosis. Chest films made in April, 1950 (Fig. 4, *A*), demonstrated the cavity in the left upper lobe which contained a fluid level in the region of the fifth intercostal space posteriorly. A later film in May, 1950 (Fig. 4, *B*), demonstrated the cavity empty of its liquid contents and a small collection of "sand" in the dependent portion of the cavity. Laminogram of the left lung (5 cm. level) (Fig. 5) revealed cavernoliths clearly at a time when the cavity was completely filled with secretion.



A. *B.*

Fig. 4.—*A*, Roentgenogram (Case 3) made in April, 1950, demonstrating a fibrotic process in the left upper lobe with cavitation. In the region of the fifth interspace posteriorly a fluid level is demonstrated in the cavity and collection of calcium is near the base of the cavity.

B, Roentgenogram (Case 3) made in May, 1950, which demonstrates the cavity in the left upper lobe empty of fluid. The calcifications are strongly suspicious of cavernoliths.

On Dec. 4, 1950, a left apical posterior segmental resection was performed* and in ten days was followed by a partial left upper thoracoplasty. Recovery was uneventful. In the resected segment there was a cavity measuring 3.5 cm. in diameter which contained about 20 c.c. of thin pus and multiple granular cavernoliths.

CASE 4.—W. E. M., a 49-year-old man, was found to have pulmonary tuberculosis in 1947, at which time the sputum was positive for mycobacterium tuberculosis, and roentgenograms of the chest revealed extensive cavitary disease of the right lung (Fig. 6). With a combination of sanatorium care and controlled bed rest at home the lesions remained stationary.

*By Mr. Brock.

In February, 1950, the patient became acutely ill with malaise, fever, and leukocytosis and there was fresh pneumonic infiltration in the right lung (Fig. 7). It was noted that the calcifications in the region of the cavity in previous films (Fig. 6) had changed position relative to each other. The pneumonic episode responded to bed rest and to para-aminosalicylic acid which was administered daily. In December, 1950, surgery was being considered but the patient was discharged from the hospital at his own request pending readmission later.



Fig. 5.—Laminogram (Case 3) measured 5 cm. from patient's back, demonstrating the abscess filled with secretions. The appearance of the calcifications in the base of the cavity confirmed the suspicion of cavernolithiasis from films in Fig. 4.

CASE 5.—Mrs. I. G., a 24-year-old housewife, was discovered to have pulmonary tuberculosis by mass radiography in 1943. Gastric washings were positive for acid-fast bacilli and there was scattered infiltration in both upper lung fields on roentgenography. Pneumoperitoneum and temporary left phrenic crush were instituted, and the pneumoperitoneum was maintained for the ensuing five years with partially satisfactory results. Laminograms (Fig. 8, *A* and *B*) of the left upper lobe made in November, 1950, revealed a cavity in the posterior apical segment of the left upper lobe and the cavity contained multiple cavernoliths. At this time the sputum was again positive.

On Dec. 8, 1950, a segmental resection of the left upper lobe was done removing the entire cavity, and the patient recovered uneventfully. The resected specimen contained a tuberculous cavity measuring 3 by 2 by 1.5 cm. in diameter, and there were several small cavernoliths lying free in the cavity.

CASE 6.—M. A., a 22-year-old housewife, was demonstrated to have had pulmonary lesions of tuberculosis at the age of 10 years, after her mother had died of the same disease. After six months of bed rest, she had resumed normal activity until she was 18 years of age, when roentgenograms revealed fresh infiltration in the apices of both lungs. She was admitted to a



Fig. 6.



Fig. 7.

Fig. 6.—Roentgenogram of chest (Case 4) demonstrating extensive tuberculous involvement of the right upper and lower lobes with multiple cavitation. In the cavity revealed in the region of the seventh interspace posteriorly several calcifications of varying size are visible.

Fig. 7.—Roentgenogram of chest (Case 4) made in February, 1950, after an acute illness. Compared to the findings demonstrated in Fig. 6 in the same patient there is evidence of recent pneumonic involvement on the right. The change in position of the calcium establishes the diagnosis of cavernolithiasis.

sanatorium where gastric washings revealed mycobacterium tuberculosis. During the eighteen months she remained in the sanatorium gastric samples were intermittently positive. At the time of discharge in May, 1950, however, the samples had been negative for three months. Her entire illness was asymptomatic.

She remained bacteriologically negative for the ensuing two years. Early in 1951 she became pregnant, and culture of gastric contents was positive in April, June, and August of that year. She remained on a modified bed rest program and had a normal delivery in October, 1951.

Roentgenograms consistently revealed bilateral infiltrations of the lung, but the lesion in the left apical region was of particular interest. Roentgenograms in the lordotic position made in March, 1951 (Fig. 9), revealed a cavity in the left upper lobe with adjacent calcification. In October, 1951, the cavity had filled with secretions (Fig. 10). It was reasoned that the calcifications which had changed position repeatedly were blocking the communicating bronchus. Review of the films made in the past several years indicated that the sequence of blocking and emptying had occurred repeatedly.

On Dec. 20, 1951, a segmental resection of the left posterior superior segment of the left upper lobe was performed removing the cavity. Recovery was uneventful. When the specimen was incised the cavity, full of thin pus, was encountered (Fig. 11). It contained a half dozen cavernoliths measuring up to 4 mm. in diameter.



A. *B.*

Fig. 8.—*A* and *B*, Laminograms (Case 5) made 5 and 6 cm. respectively from the back, demonstrating an open cavity in the apex of the left upper lobe and at least three cavernoliths of different sizes.



Fig. 9.



Fig. 10.

Fig. 9.—Roentgenogram (Case 6) made in March, 1951, in the lordotic position to visualize the apex of the left upper lobe and demonstrating an open cavity in the region of the third intercostal space posteriorly. Calcifications are not well demonstrated in this photograph. Sputum was positive for mycobacterium tuberculosis.

Fig. 10.—Roentgenogram (Case 6) made in October, 1951, demonstrating the abscess cavity previously revealed in Fig. 9, which is now completely filled with secretions. There are multiple calcified bodies visible in the base of the cavity. Sputum and gastric washings contained no acid-fast organisms.

DISCUSSION

Analysis of this small group of patients reveals no symptomatic pattern which is characteristic of cavernolithiasis. Brief comment, however, is due certain clinical aspects of the condition which are demonstrated in these six patients and which may obtain diagnostic significance as experience with cavernolithiasis increases. For the most part the cavernolith is a "silent stone" and produces no symptoms (Cases 3 to 6). It appears, however, that the calculus may pro-



Fig. 11.—Photograph of the sectioned specimen of left upper lobe (Case 6) demonstrating the abscess cavity which contained thin pus. In the lesion are multiple cavernoliths measuring up to 4 mm. in diameter.

duce symptoms either by local trauma and irritation of the pulmonary parenchyma (severe cough, hemoptysis, etc.) or by interference with drainage of the cavity (fever, malaise, pleuritic pain, etc.). Case 2 is a striking example of the marked pneumonic symptoms which may occur as a result of acute obstruction to cavitary drainage. The pneumonic episodes in Case 4 may also have been a manifestation of a blocked cavity. The most marked symptoms suggestive of pulmonary irritation were demonstrated in Case 1. In this instance collapsing the cavity by first stage thoracoplasty probably caused the sharp cavernolith to lacerate a pulmonary vessel producing a massive hemoptysis and dissemination of the disease to previously uninvolved lobes. In contrast to the broncholith,

the cavernolith is rarely coughed up. In Case 2 a small calculus passed through the communicating bronchus, but usually the small caliber of the opening compared to the size of the cavernolith favors impaction of the stone and cavitary obstruction.

Even though the course of the tuberculous process was in some instances altered by the presence of intracavitary calculi, diagnosis usually depends upon roentgenographic findings. Demonstration in a single chest film of calcification in the peripheral lung fields in the neighborhood of a cavity should arouse suspicion that the calcium is lying inside of a cavity. The precise diagnosis of cavernolithiasis, however, depends upon demonstrating a change of position of the calcified material by careful examination of multiple roentgenograms. Ordinarily the cavernoliths occupy a dependent position in the cavity but sometimes may adhere to the walls of the cavity in any position. Therefore, films made in the recumbent positions as well as the erect may assist in demonstrating the migration of the fragment. Other techniques including induced coughing may be helpful in dislodging the calculus and moving it to another position in the cavity. The most valuable technique for demonstrating the cavernolith is laminography. Figs. 5 and 8 are examples of laminography in these cases.

In my opinion therapeutic management of patients with cavernolithiasis differs from that of the usual patient with cavitary tuberculosis and herein lies the necessity for establishing a precise diagnosis. Inert substances lying in infected spaces anywhere in the body interfere with natural healing processes, and in none of these cases of cavernolithiasis did spontaneous healing of a cavity occur. Expectoration of a piece of a calculus occurred in Case 2 but several larger ones remained in the cavity. It may be concluded, therefore, that surgical removal of the lesion is the logical means of obtaining a cure. Techniques of pulmonary collapse including pneumothorax, thoracoplasty, and plombage are theoretically unsound in cavernolithiasis. In the only case where thoracoplasty was employed, near fatal consequences ensued from massive hemoptysis. Collapse procedures may also produce acute cavitary obstruction with possible perforation from overdistention. As already mentioned, severe pneumonic manifestations may accompany such cavitary obstruction. Although many patients in the past must have been successfully treated by collapse techniques, it appears on theoretical and clinical grounds that partial pulmonary resection provides the most rational method of treatment. In five patients so treated the final result was satisfactory.

SUMMARY

Attention is directed to a specific type of pneumolith encountered within pulmonary cavities of tuberculous origin. To these calculi the term cavernolith is applied.

Six patients with cavernolithiasis are reported briefly. In most instances, the stones were silent but occasionally irritative, and obstructive phenomena significantly altered the clinical course.

Diagnosis is rarely made on clinical findings and is usually based upon roentgenography. Careful examination of serial films throughout the clinical

course with particular attention to possible variation in position of intrapulmonary calcification is essential. Laminograms are useful in demonstrating the intracavitary position of such concretions.

Cavernolithiasis is an indication for surgical intervention in pulmonary tuberculosis, and pulmonary resection is recommended in preference to collapse procedures.

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INDICATIONS FOR THORACOPLASTY AND PULMONARY RESECTION IN THE SUPINE POSITION

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CHARLOTTE, N. C.

DURING the past two years we have adopted the supine position in certain cases in which pulmonary resection and thoracoplasty were combined as a one-stage procedure. In this position, proper ventilation of the lungs is carried on more automatically and spontaneously; thus the patient is less likely to develop respiratory acidosis. We prefer this position particularly in resections for tuberculosis when pleural symphysis exists, when previous thoracoplasty has been performed, when the patients are acutely ill, and when the vital capacity is low.

If one or more of these conditions exist, the advantages provided by the anterior approach over the commonly employed posterolateral are several. The inclusion of a small anterior thoracoplasty with the pulmonary resection allows a partial collapse of the chest wall which is often highly desirable in resections for tuberculosis. Overexpansion of the remaining lung in such instances may lead to reactivation or spread to the contralateral lung in tuberculosis, and may lead to emphysema after pneumonectomy.

The question may arise as to the ability of an anterior thoracoplasty to produce sufficient thoracic cage collapse and thereby prevent overdistention of the remaining pulmonary alveoli. In our small series of thirty-one resections, the collapse obtained through this procedure appeared adequate to prevent overdistention of the remaining lung.

Any individual who after a two- or three-rib thoracoplasty later requires lobectomy will usually develop atelectasis if a rib is removed in the conventional posterolateral area. In such a situation we have found this procedure particularly indicated. Furthermore, if thoracoplasty has been performed previously the anterior approach is simpler, safer, and preferable. Here, the anterior intact rib stumps and cartilages simplify the problem of finding a cleavage plane; thus a tedious and bloody dissection through a regenerated bony plate posteriorly is averted. Likewise, the normal anatomy of the anterior mediastinum spares the surgeon the difficult problem of creating and establishing a cleavage plane through adherent and dense pleura. The problem is made even simpler since acid-fast lesions are rarely situated anteriorly. Minimal adhesions are produced on a free pleura anteriorly and medially through which the mediastinal structures are rapidly exposed and rapidly identified. In addition, this anterior exposure diminishes the major hazard of excessive blood loss. This is accomplished by controlling and ligating the pulmonary vessels before attempting to extirpate adherent, fibrotic, abscessed lung from the parietal pleura or chest wall.

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Finally, the suggested method of exploration not only technically facilitates the operative procedure, but the patient is maintained in a normal physiologic position throughout surgery. This position reduces to a minimum the hazard of spilling bronchial secretions into the contralateral lung, though it permits excellent pulmonary gas exchange which may avoid dangerous accumulation of carbon dioxide in the blood. The latter problem has been brought to our attention recently by the studies of Beecher and Murphy[1] and also those of Maier.[2] It is thought that the lateral position favors the development of acidosis.

Dehiscence of the bronchus in resection for tuberculosis seemingly is a universal complaint. If a bronchial pleural fistula does develop the situation is not too grim, since already a partial thoracoplasty has been performed. Furthermore, the cavity, which is under tremendous pressure, can be readily decompressed by simply inserting a catheter; or better still, an incision can be made through the soft tissue of the anterior chest wall and the cavity packed with gauze. Hereby, the contamination of the contralateral lung has been eliminated by the gauze occluding the fistula. Then with safety the patient can be turned to the lateral position for completion of the thoracoplasty.

The only disadvantage of the procedure is the greater deformity which follows an anterior thoracoplasty. However, the efficiency and safety of this surgical undertaking, and the preservation of shoulder function must also be of concern to the surgeon. The majority of these patients are in younger age groups in which a long life expectancy is in order. The shoulder muscles are not interrupted or disturbed with this operation. Such an advantage will more than compensate for the deformity of anterior depression which can be readily camouflaged.

OPERATIVE TECHNIQUE

A curved skin incision is made, beginning at the opposite third costal cartilage and extending across the sternum under the mammary gland to the posterior axillary line (Fig. 1). The superior skin flap, with the breast, is dissected free from the pectoralis fascia up to the clavicle (Fig. 2). Unless a long skin incision is used and the flap reflected above the clavicle, the surgeon cannot obtain adequate exposure of the upper ribs and cartilages. The pectoralis major is divided longitudinally about 1 to 2 cm. from the sternum, divided transversely at the fifth costal cartilage, and then retracted laterally (Fig. 3). The second to fifth ribs, inclusively, are removed subperiosteally extending to the posterior axillary line together with their corresponding cartilages (Fig. 4).

Frequently the anterior two-thirds of the first rib is also resected, especially when the inflammatory process has attached the lung tenaciously to the apical pleura. We have experienced no injury to the subclavian vessels in removing the first rib. The pleural space is usually entered through the bed of the third rib, then the mediastinal pleura is exposed and incised. The first vessel encountered in the mediastinum is doubly ligated, transfixed, and severed. The remaining structures are isolated in accordance with the surgeon's choice. Lastly, it is usually the bronchus that is clamped and transected. The lung is then detached from its adherent adjacent structures, largely by sharp and blunt

Fig. 1.

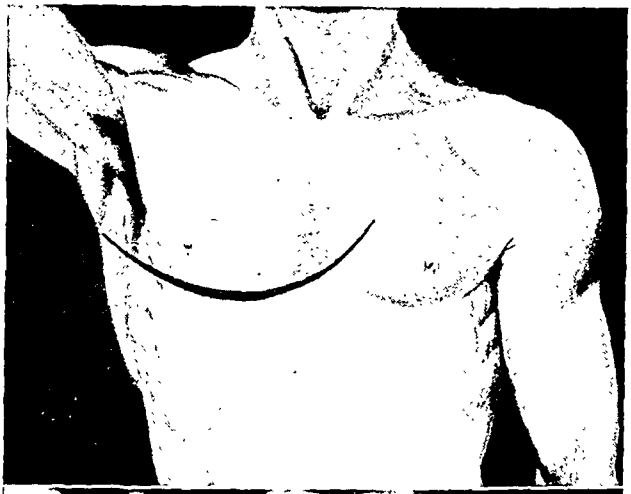


Fig. 2.

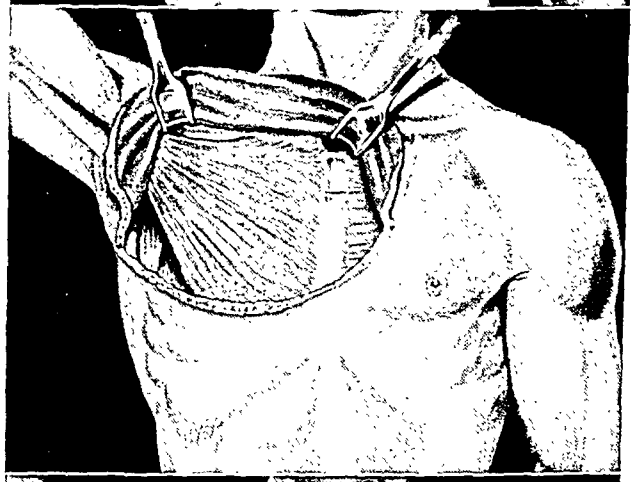


Fig. 3.

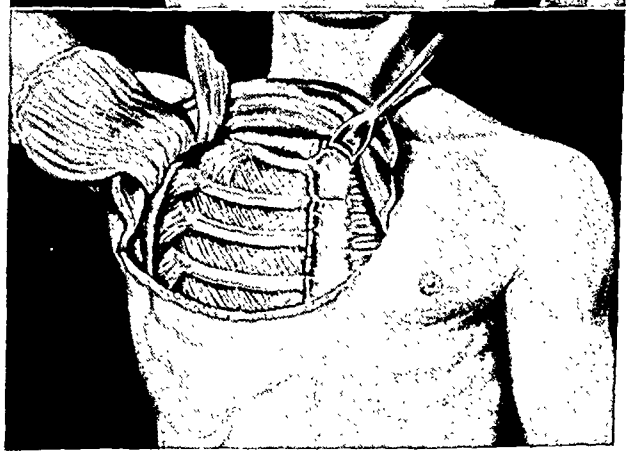


Fig. 1.—A long skin incision beginning at the opposite costal cartilage is extended to the posteroaxillary line.

Fig. 2.—The skin flap is reflected above the clavicle.

Fig. 3.—The pectoralis major muscles are divided and retracted.

dissection which on occasion may require strong arm avulsion. Chest closure is simple since no attempt is made to approximate the pleural or intercostal bundles. It is accomplished by suturing the pectoral muscles, the subcutaneous tissue, and the skin. A frail chest wall has not been a problem. The chest is collapsed stably with abdominal pads held in place by adhesive tape applied longitudinally.

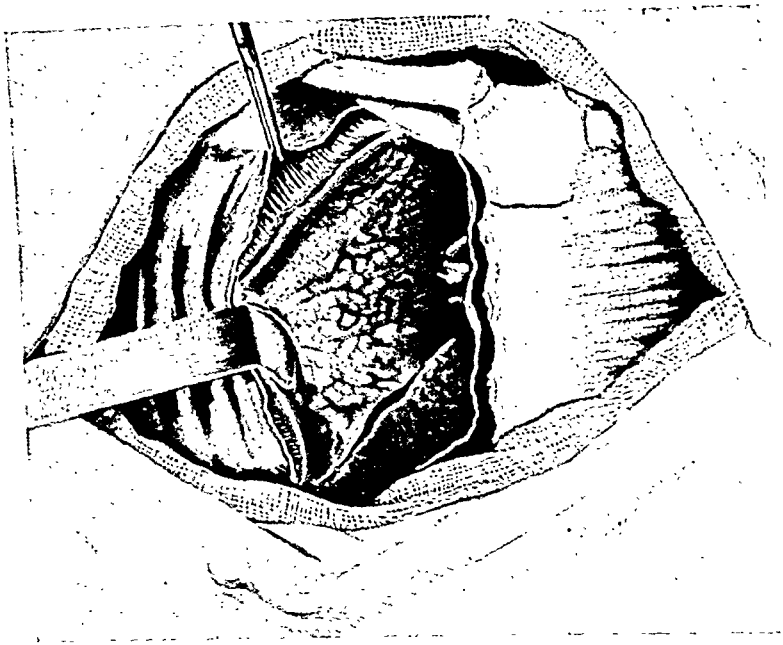


Fig. 4.—The first five cartilages and ribs have been removed as far posteriorly as the posteroaxillary line.

From a technical aspect several points should be emphasized. Following anterior rib resection, the major vessels and mediastinal structures are readily accessible and easily isolated and controlled. In contrast, the patient operated upon in the lateral position has his mediastinal structures "at the bottom of the well." Moreover, if intrapericardial exposure of the vessels becomes necessary, the anterior route lends itself admirably for such an eventuality.

The following case reports of patients, operated upon in the supine position, illustrate a one-stage procedure for thoracoplasty and resection:

CASE REPORTS

CASE 1.—A 28-year-old white man was first admitted to Mecklenburg County Sanatorium eight years ago, complaining of severe headaches, chills, and fever. X-ray examination showed cavity formation, and a diagnosis of tuberculosis was confirmed. He remained one year at the Sanatorium, but was readmitted on three other occasions. Each time he was discharged as apparently arrested. During the third admission a right-sided empyema was drained. Since the left lung had remained stable for eight years, the patient was prepared for pneumonectomy with streptomycin and para-aminosalicylic acid. The bronchus which showed ulceration responded to chemotherapy, and surgery was undertaken.

With the patient in a supine position a curved incision was made which extended from the left third interspace, across the sternum, beneath the right breast to the right posterior axillary line. The right third rib was identified and resected subperiosteally, and then the costal cartilage was removed subperichondrially. The fourth, fifth, and second ribs and cartilages were similarly removed as far as the posterior axillary line. Because of the markedly thickened and adherent pleura, separation of the lung from the lateral and posterior chest wall was impossible. In this circumstance the mediastinum was attacked first. The elements of the hilum were treated by individual ligation technique, and then with considerable difficulty and much bleeding, the lung was bluntly torn from the parietal pleura. Bleeding was controlled with pressure, and the chest wall was closed in layers.

All vital signs remained good, and no complications arose. The patient is now in good health and has been working for eighteen months.

CASE 2.—This 41-year-old white woman had been admitted to the Mecklenburg Sanatorium on four occasions since 1937 because of reactivation of a left pulmonary tuberculosis and empyema. During the last stay (nine months) in spite of streptomycin and PAS, the sputum remained positive. The trachea and mediastinum were deviated to the diseased side. Roentgenographic and bronchoscopic findings limited the pathology to the left lung. A pneumonectomy was performed as described in Case 1. The lung was firmly adherent to the chest wall and diaphragm. Because of the adherence of the lung to the parietal pleura, there was a large amount of bleeding upon avulsing it from its attachments. This bleeding was readily controlled with packs.

Convalescence was without incidence until the fourth month after the operation when she developed signs and symptoms of a bronchial pleural fistula. This complication was managed by inserting a No. 22 catheter into the distended soft tissue in the midclavicular line. The cough was lessened but not controlled until a transverse incision 6 to 8 cm. in length was made through the thin anterior chest wall and the cavity packed with gauze.

A few days later, with the patient placed in the posterolateral position, a seven-rib thoracoplasty was done. The gauze packing prevented any spillage into the opposite bronchus during the operation. The cavity was loosely packed as often as indicated, until three months following the thoracoplasty when only a small fistula remained; but this finally ceased to drain and whistle. One year after pneumonectomy the patient was discharged without any sequelae.

SUMMARY

In certain instances the anterior approach for pulmonary resection is preferable to the posterolateral one because:

1. In the supine position fatal acidosis may be avoided because of better pulmonary ventilation.
2. The incidence of contralateral infection is decreased.
3. It permits a combined thoracoplasty and pulmonary resection in one stage with minimal shock.
4. Cleavage planes in the chest wall and mediastinum are readily obtained in cases where thoracoplasty was previously performed.
5. Mediastinal structures are accessible for ligation prior to the separation of the lung from the chest wall, thus minimizing blood loss.
6. If a bronchial pleural fistula does develop, its consequence is less severe and its correction is more easily accomplished.

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CARCINOSARCOMA OF THE ESOPHAGUS

Report of a Case With Autopsy Findings

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THE infrequent neoplasms exhibiting more than one cellular component provide interesting material for study and provoke considerable speculation as to their origin. Such a tumor was encountered in the esophagus as an incidental finding in a patient dying of pulmonary tuberculosis.

CASE REPORT

J. K., a Polish man, aged 61 years, was admitted to the Municipal Tuberculosis Sanitarium with a history of having been hospitalized approximately ten years before, in another institution, for pulmonary tuberculosis. He left that institution against medical advice after a four-year stay and worked at odd jobs until the time of his admission here. His family history was negative except for the fact that his mother died at 40 years of age of some "stomach disorder." This man worked at various labor jobs all of his life, did not use tobacco, but was a moderate drinker. Venereal disease was denied. The onset of illness was characterized by a cold with a nonproductive cough, temperature up to 100° F., and a weight loss of thirty pounds. A roentgenogram of the chest and the presence of acid-fast bacilli in the sputum confirmed the diagnosis of pulmonary tuberculosis at a sanitarium dispensary.

Clinical Findings.—Physical examination at the time of admission revealed a tall, fairly well-nourished white man who had several carious teeth and evidence of pyorrhea. The abdomen was slightly protruding but no organs or masses could be palpated. Percussion of the chest revealed some dullness over both upper lung fields, and an occasional râle was heard.

Course.—Approximately two years after admission to the Sanitarium he complained of some difficulty in swallowing. Examination by an otolaryngologist at this time revealed a mass in the right laryngeal ventricle, which, on biopsy, proved to be a papilloma containing some small tubercles. Also, a tumor mass was noted in the mucous membrane overlying the medial surface of the right mandible opposite the first to third molar teeth. This lesion was ulcerated with folded edges, and measured 1.0 by 2.5 cm.; a biopsy proved it to be an epidermoid carcinoma. The laryngeal tumor was excised, the mandibular tumor was treated by x-ray radiation, and the patient's dysphagia gradually disappeared. It is of interest that the painful swallowing the patient complained of was limited to the mouth and throat region. In spite of supportive therapy the patient's pulmonary disease progressed, and he died approximately three years after admission.

Pathology.—At autopsy, examination of the buccal cavity revealed a smooth mucous membrane. No gross abnormality could be seen in the pharynx or larynx. The right lung was firmly adherent to the chest wall over the apical portion by dense fibrous adhesions; the left lung was adherent to the chest wall by less dense adhesions. On sectioning the lungs, a cavity was found in the posterior aspect of the right upper lobe. This cavity measured 6 cm. in greatest dimension and was surrounded by considerable scar tissue. In the opposite lung numerous acinous-nodose lesions were seen, and there was considerable congestion of the uninvolved lung parenchyma. The major bronchi showed considerable congestion. Upon opening the esophagus a firm, pedunculated, smooth, yellow-gray, kidney-shaped tumor which measured 5.0 by 2.5 by 1.5 cm. was exposed (Figs. 1 and 2). This

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neoplasm was attached to the mucosa 9 cm. below the larynx by a thin pedicle. Its surface was in part smooth and in part granular in appearance. On surfaces made by sectioning it was pale gray to yellow-gray in color and resembled a soft fibroma.

Microscopic examination of the esophageal tumor mass revealed that there were two different cellular constituents. The most striking feature was the sarcomatous nature of the stroma which displayed many huge bizarre giant cells and appeared to be actively proliferating (Figs. 3 and 4). In addition, there were nests or groups of epithelial cells which, for the most part, were undifferentiated but which in areas showed some tendency toward rosette formation (Fig. 5). In a preliminary survey of the sections, the impression was given that this might be of neurogenous origin because of the presence of some cells which had the appearance of atypical ganglion cells, and small groups of cells not unlike neuroblasts or sympathicoblasts; however, the consensus of opinion of several consultants[1] was that this represented a carcinosarcoma rather than a tumor of neurogenous origin. Several typical epithelioid tubercles were noted near the periphery of the tumor.

Microscopic studies of the buccal mucosa revealed only moderate fibrosis attributable to the x-ray therapy which had been given approximately seven months previously.

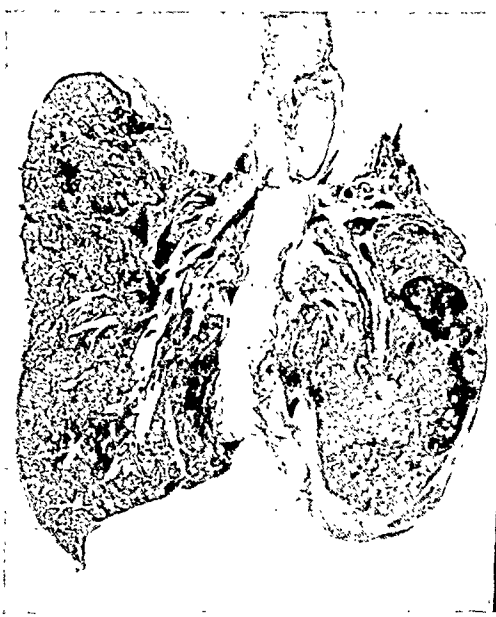


Fig. 1.



Fig. 2.

Fig. 1.—A photograph of the midsection of the lungs with the esophagus still attached showing a tumor mass 5 by 2.5 by 1.5 cm. located in the upper half of the esophagus at a point 9 cm. below the larynx.

Fig. 2.—Close-up of the tumor mass, demonstrating the pedicle, smooth glistening surface, and lack of mucosal involvement.

DISCUSSION

As early as 1904, Hansemann expressed the opinion that the term carcinoma sarcomatoides should be reserved for the description of a carcinoma with a sarcomatous stroma. He supposedly observed one such case in the esophagus but no histopathologic description was recorded.

Herxheimer, in 1908, characterized carcinoma sarcomatoides as being composed of two distinct cellular types—flat epithelial masses within or surrounded by a sarcomatous stroma.

Fig. 3.

Fig. 4.

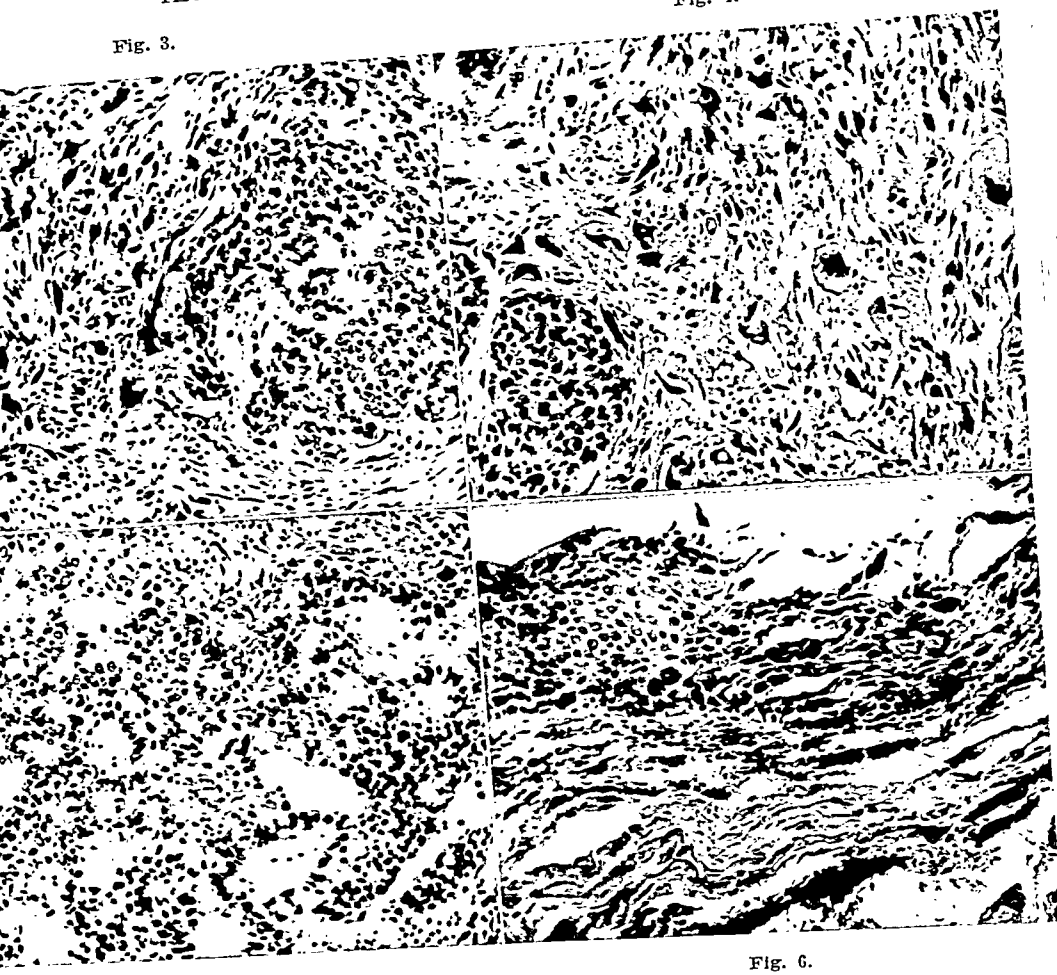


Fig. 5.

Fig. 6.

Fig. 3.—Photomicrograph of the tumor mass showing groups of undifferentiated epithelial cells surrounded by a sarcomatous stroma. The stroma shows many bizarre giant cell forms. (Hematoxylin and eosin stain, ×150.)

Fig. 4.—Photomicrograph of a different area showing a similar histologic picture. (Hematoxylin and eosin stain, ×150.)

Fig. 5.—Photomicrograph of an area demonstrating pseudo-rosettes. (Hematoxylin and eosin stain, ×150.)

Fig. 6.—Photomicrograph of the esophageal mucosa at the base of the pedicle demonstrating some activity in the mucosa and an intact basement membrane. (Hematoxylin and eosin stain, ×150.)

In an excellent review Saphir and Bass[2] pointed out that the carcinosarcomatous nature of 153 so-called carcinosarcomas was very questionable. Careful re-examination of sections from tumors thought to be carcinosarcomas revealed that the majority of the neoplasms were probably primary carcinomas. In evaluating the sarcomatous features of the reported carcinosarcomas these authors pointed out that complicating factors may be present which play a role in the alteration of the fundamental histologic appearance of the tumors. These factors are: the spindle-shaped variation of carcinoma cells which may be interpreted as sarcomatous instead of the variant they

actually are; marked anaplasia of the carcinoma cells; chronic inflammation which may alter the morphology of the tumor cells by producing much connective tissue which may be construed as sarcomatous in nature or by provoking a lymphocytic reaction sometimes interpreted as a lymphosarcomatous component of some of these tumors; and the invasion of a benign connective tissue tumor by a carcinoma. Two years later, Pearlman[3] reviewed the cases of carcinosarcoma of the esophagus and examined in detail eight cases of malignancy of the esophagus in which autopsy had been done at Michael Reese Hospital.* These cases were studied with the question in mind as to how their histopathologic picture might be reconciled with the diagnosis of carcinosarcoma, and he concluded that the existence of so-called carcinosarcomas in the esophagus was questionable. Likewise, in 1949, Stout, Humphreys, and Rottenberg[4] reviewed the literature for carcinosarcomas of the esophagus and listed twenty cases in all. Of these twenty cases, only ten were polypoid growths exhibiting epithelial and sarcomatous elements and three of these ten cases showed definite metastases. In nine instances the lesion was in the lower half of the esophagus, in the tenth it was located in the hypopharyngeal region.

There are several interesting aspects worthy of mention in the case being presented. First, no evidence of metastases could be demonstrated, there being no invasion of the esophageal mucosa, even though some activity was noted in the epithelial layer at the site of pedicle attachment (Fig. 6). Second, this individual had two primary tumors—carcinosarcoma of the esophagus and an epidermoid carcinoma of the buccal mucosa. The multicentric origin of tumors is well known, and certainly from a histologic and anatomic standpoint there is no connection between these two neoplasms. Third, this growth was an incidental finding at autopsy, there being no clinical symptomatology or x-ray evidence of the neoplasm prior to death. Inasmuch as the man was extremely ill from pulmonary tuberculosis it is very possible that this masked any symptoms of the lesion, as is so often the case. Last, another feature of this case, not illustrated, was the presence of several early tubercles about the periphery of the tumor mass. Normal esophageal mucosa is rarely, if ever, involved in a tuberculous inflammatory process.

SUMMARY

A case of carcinosarcoma of the esophagus is reported. The pedunculated nature and location of the tumor are illustrated as well as the dual histologic pattern. This was an incidental finding in a case of pulmonary tuberculosis and epidermoid carcinoma of the buccal mucosa.

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LYMPHATIC DRAINAGE FOLLOWING INTRABRONCHIAL INSTILLATION OF SILVER-COATED RADIOACTIVE GOLD COLLOIDS IN THERAPEUTIC QUANTITIES

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INTRODUCTION

THE use of radioactive metallic colloids promises at the present time to become a useful adjunct to surgery in the treatment of bronchiogenic tumor in man. Recent reports concerning lung cancer in a relatively large series of patients are in surprising agreement in three important particulars,[1, 2, 12, 13] namely: (1) bronchiogenic or primary carcinoma of the lung is diagnosed approximately five to six times more commonly than a generation ago; (2) in only about one-fourth to one-third of the patients are the tumors resectable when first seen; (3) the over-all "five-year cure rate" is in the neighborhood of 5 to 7 per cent. At least two of these groups, Oschner and DeBakey, and Evarts Graham in St. Louis contend that this tumor is now more common in males than carcinoma of the stomach which previously occupied first place. The importance of these statistics cannot be overemphasized and should have quite an impact on the medical profession.

Subject to early diagnosis the treatment of choice in this condition is apparently pneumonectomy, although a few surgeons prefer lobectomy. However, this procedure is applicable only in approximately one-fourth of all the patients who present themselves for study and diagnosis. Metastases by way of the lymphatics is the usual course taken. Deep x-ray treatment may increase slightly the time of survival but by many is considered to be of relatively little value except as a palliative agent. It is not necessary to go into the difficulties involved in the irradiation of deep-seated thoracic lymph nodes, whose position can only be guessed at in any individual and whose irradiation must necessarily involve undesirable side reactions of the pleura and lung parenchyma. Therefore it would seem that any potentially useful procedure which might be used in conjunction with pneumonectomy to cut down the propagation of this tumor process should be a very acceptable one, at least from an experimental viewpoint.

In previous reports we have shown that when radioactive gold colloids are administered intratracheally[10] or intrabronchially[9, 10] a considerable amount of radiation of the lung parenchyma may be achieved. Under these conditions drainage by the thoracic lymphatics is relatively slow and therefore the amount of irradiation to the site of common metastases is limited. Later it was demonstrated that radioactive metallic silver colloids were drained much more rapidly

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by the lymphatics.[6] Their use on a therapeutic scale, however, is obviated at the present time by difficulties in technical production. Ag^{110} colloids, although useful experimentally, are not suitable for therapeutic use due to the long life of this material (225 days half life) which would probably render them carcinogenic in themselves. Ag^{111} is produced by a transmutation reaction $Pd^{110} + n \longrightarrow Pd^{111} + y$, the latter by beta decay being converted to Ag^{111} with a half life of 7.5 days. However the cross section of the Pd to thermal neutrons is relatively low and the abundance of the parent Pd^{110} is only 13 per cent. As a result units obtained from the Oak Ridge pile yield only about 5 mc. of the radioactive silver isotope and these would probably not be sufficient for therapeutic trial. It is planned that in the near future instead of 1 Gm. of Pd being irradiated that the metal in quantities of perhaps 5 Gm. be subjected to thermal neutrons in a higher flux pile. This should make possible activities of about twenty times those now obtainable.

In the meantime we have shown that if radioactive gold colloids are used as nuclei, the dispersion of silver results in a silver-coated colloid which behaves in the same manner physiologically as the silver metal colloids when introduced by bronchoscope. Using such coated colloids in tracer quantities it was found that appreciable amounts were drained by the thoracic lymphatics within from two to five days following administration.[4]

According to the present methods used, there appears to be a minimal amount of silver required to coat such gold nuclei and therefore total radioactivities due to the gold are limited somewhat due to the possible toxic effects of large amounts of silver. In order to obviate this in attempts to use therapeutic quantities of the radioactive gold, bombardments of Au^{197} at the Brookhaven National Laboratories were carried out with resultant specific activities of approximately five times those obtainable previously in the Oak Ridge pile. Following the usual processing* these materials were coated as will be described and administered by bronchoscope using an 18 gauge inner metal tube, the proximal end of which was fitted for a syringe† for injection into the desired lobe of the lung.

The earlier work with the straight gold colloids introduced by this route, in a fairly large series of dogs, indicated that the tolerance dosage was approximately 0.5 to 0.7 mc. per kilogram of body weight, higher dosages usually resulting in death of the animal apparently due to pulmonary inflammatory processes and their sequellae.[9] In contrast, dogs to which the silver-coated colloids have been administered in quantities as high as ten times the amounts just stated have failed to show any untoward clinical manifestations. Although at autopsy some of the latter animals showed considerable degrees of irreversible pathologic changes in the lung lobe involved, nevertheless they, for periods of from one week to several months, exhibited no untoward symptomatology. It is felt that in all likelihood the silver coating, in promoting relatively rapid transfer from the lung parenchyma to the lymphatic system, thus protects the lung from an inordinate amount of irradiation over a relatively prolonged period of time.

*This was done by Abbott Laboratories, North Chicago, Ill.
†Manufactured by Eisle Corporation, Nashville, Tenn.

It seems quite likely that this effect is achieved by reason of an irritation phenomenon, occasioned by the presence of silver which is gradually leached off the colloid, giving rise to enhanced lymphatic drainage. Grossly the pathologic changes in the lung were much less than produced by the gold colloids. Histopathologically it was found in many instances that the lymph node architecture was completely abolished, complete hyalinization having occurred, and in other nodes considerable alteration of the structure having taken place.

Within the past year there has been reported by Muller and Rossier[11] another procedure for irradiation of the lung parenchyma. These investigators employed the microembolic phenomenon obtainable by the introduction of large gold particles introduced intravenously through cardiac catheterization. In an elaboration of their technique they adsorbed soluble gold onto relatively uniform particles of carbon of approximately 30 to 50 micra in size, following which the gold was reduced to the metallic form and subjected to irradiation by thermal neutrons. This latter procedure minimized the amount of small particulate matter which would be carried through the capillary bed and on to the reticuloendothelial system. It is to be stressed that these particles are of about one thousand times the size of those used in the investigations reported by ourselves. The previously mentioned authors show radioautographic evidence of uniform distribution of the radioactive material throughout the lung. Unfortunately we do not have any similar type of evidence for such distribution in our animal work described in this presentation. However, there is every reason to believe that whether the microembolic procedure or the intrabronchial insufflation method is used that the lung parenchyma will be adequately irradiated, in the first case from the capillary bed and in the latter case from the alveoli. It must be kept in mind, however, that most bronchiogenic tumors derive their blood supply from the bronchial artery, and therefore one gains very little in attempting to irradiate the tumor mass proper or the lung parenchyma by either of these procedures. It is especially to be noted that introduction of the large particle gold by the blood vessel route does not result in drainage by the lymph nodes of the thoracic cavity which would seem to be the most important contribution to the use of radiation as an adjunct in surgery as provided by radioactive isotopes.

METHODS

A typical shipment obtained from a Brookhaven bombardment through Abbott Laboratories, as received, consisted of 6.5 ml. of gold colloid containing 0.6 mg. of metallic gold per milliliter and a radioactivity amounting to 50 mc. per milliliter. Left in its original shipping vial, 2 ml. of silver nitrate solution containing 25 mg. of silver was injected through the rubber stopper following which 100 mg. of ascorbic acid dissolved in 2 ml. of pyrogen-free sterile distilled water was added. Actually there is some dispersion of the silver following the addition of the silver itself due to an excess of ascorbic acid which is present in the original gold colloid preparation. The addition of the extra and excess quantity of ascorbic acid results in the production of a deep brown colloid which on high dilution is brown in color to reflected light and a yellowish gray

to transmitted light. Occasionally it was found that the development of color required that the preparation be allowed to stand for as much as an hour. The vial was then reautoclaved and taking into consideration the volume dilution the radioactivity per milliliter was recalculated and the material used for treatment.

Animals were dewormed, vaccinated against distemper, and maintained in the laboratory for a number of weeks prior to use in order that their physical condition would be good. They were anesthesized with pentobarbital sodium (35 mg. per kilogram) and strapped to a table whose upper end was raised somewhat to facilitate drainage downward into the lung. A small-sized bronchoscope was inserted, usually into the right lower middle lobe. An 18 gauge inner metal tube which had been fitted for a syringe was then inserted. A lock-type syringe was attached and the colloid was injected as rapidly as moderate pressure on the plunger would allow. Carried out in open air this results in a stream of colloid emerging about 3 to 4 inches beyond the end of the inner tube and is therefore thought to result in the administration of the material under turbulent conditions which may facilitate the uniform distribution of the colloid in the alveoli of the insufflated lobe. The animal was laid on the floor on his right side until recovery from the anesthesia occurred. In no instance was there any evidence of coughing noted. In these animals the volume of material insufflated was maintained at 5 ml. As stated previously in earlier experiments[4, 9, 10] the colloid was administered at a level of 0.5 ml. per kilogram, but it is not felt at the present time that the optimal volume dosage has been ascertained.

In several of the experiments attempts were made to trace the relative mobility of the radioactive silver (Ag^{110}) coated gold nucleus (Au^{198}) colloid, while in others the material insufflated consisted in an ordinary silver coat on the highly specific radioactive gold nucleus where histologic evidence of damage to the lymph nodes was of special interest. Where the coating and nucleus were both radioactive, samples were counted with a standard commercial beta counter at intervals of approximately one week, weighed fresh tissues being used and stored under refrigeration between counting. In this way radioactivities were measured throughout the dissipation of the radioactivity of the Au^{198} allowing for final determination of Ag^{110} activities and determination by difference of the Au^{198} activities by subtraction of the final from the original measurements. The half lives of these two isotopes being 225 and 2.7 days, respectively, this was relatively simple.

The animals were usually sacrificed by the intravenous administration of an excess of nembutal. All discernible lymph nodes in the thorax were removed for study. Perinodular fat was dissected away and the nodes weighed to the nearest 5 mg. Radioactivity was determined by direct counting using a thin mica-end window counter. Where activities were very high a copper absorber was interposed. The nodes were then placed in 10 per cent formalin for subsequent sectioning and histologic study.

EXPERIMENTAL OBSERVATIONS

Prior to the use of therapeutic quantities of colloids some preliminary studies were carried out in a few dogs in an attempt to follow the courses taken,

respectively, by the silver coatings and the gold nuclei by making use of doubly tagged colloids in which Ag^{110} was employed as the coat for Au^{198} nuclei. A group of four dogs ranging in weight from seventeen to twenty-one pounds were administered Ag^{110} coated Au^{198} colloids in quantities representing approximately 4 mc. of the gold nucleus and only tracer quantities in the silver coat. These animals were sacrificed at five, eight, and twelve days and six months, respectively. Radioactivity measurements in the first three (51-93, 51-94, and 51-96) indicated that the $\frac{\text{gold}}{\text{silver}}$ isotope ratios were such as to show that some of the silver was gradually lost from the lung during the first twelve days and tended to accumulate in the liver. It is of some interest to note that the animal 51-100, sacrificed at six months, still showed considerable activities in a number of the lymph nodes as well as the lungs, especially the treated lobe, in contrast to some animals to be described later which received what is now looked upon as therapeutic quantities of coated colloids. Unlike the gold which is practically insoluble in body fluids, the silver is slowly dissolved and in such form is thought to be highly inflammatory in nature. It is felt that this perhaps constitutes the chief reason why drainage by the lymphatics is facilitated.

The next three dogs 51-71, 51-72, and 51-73 were litter mate female hound mongrels of approximately 7 months of age and weighing sixteen to twenty pounds. They were nembutalized, the bronchoscope inserted, and Ag^{110} coated Au^{198} colloids introduced into a single lobe of the lung. The activity of the silver coat was of tracer orders of magnitude but the activity of the gold nucleus in each case was 50 mc. Earlier studies[9] indicated that the tolerance of the dog to straight gold colloids administered by this route was approximately ½ mc. per kilogram of body weight. Higher doses resulted in death, usually due to pulmonary causes, within ten days to two weeks. These three animals were followed for about a period of six weeks during which time they were in excellent clinical condition and manifested no reaction to the radioactive materials. They were sacrificed by the administration of intravenous chloroform. At autopsy 51-71 showed no necrotic area in the right lung which would correspond to the site of insufflation. There were two grayish white streaks of approximately 5 to 6 cm. in length and about ½ cm. in width on the right lower lobe which it was felt probably represented the site originally involved. Activity measurements (Table I) showed highly divergent quantities of residual radio silver^{110}, there being no measurable amounts of Au^{198} activity left after about sixteen half lives of this isotope. It was interesting to note that there was 50 per cent higher concentration of radioactivity in the left mediastinal as opposed to the right mediastinal node. Similarly, the right bronchial node showed practically no radioactivity whereas nearly 300 times the concentration of this activity was found in one of the left bronchial nodes. However, in another left bronchial node there again was very little activity. Unlike many of the earlier animals given silver-coated colloids there was relatively little activity in the node at the bifurcation of the trachea. The lungs showed very little residual activities, one section, through the streaked area of the right lower lobe, however, showing a significantly higher count and indicating only that this was the site of deposition of the colloid. However, one might say that essentially

TABLE I. 51-71. INTRABRONCHIAL Ag^{110} COATED Au^{198} (50 MC.) COLLOID

	WEIGHT OF WHOLE NODE IN GM.	COUNTS PER MIN. PER GM.	COUNTS PER MIN. PER GLAND
L. mediastinal	.09	137,000	12,319
R. mediastinal	.22	99,000	21,800
L. bronchial A	.19	395	75
L. bronchial B	.05	113,000	5,601
R. bronchial	.11	625	69
R. tracheal	.04	143,000	5,025
Bifurcation	.13	6,900	900
	WEIGHT OF TISSUE SAMPLE IN GM.	COUNTS PER MIN. PER GM.	
L. upper lung	.28	189	
L. mid-lung	.37	111	
L. lower lung	.44	20	
R. upper lung	.36	120	
R. lower A (1)	.35	31	
R. lower B (2)	.63	386	
Liver	.38	540	
	Sacrificed after 37 days		

complete drainage had occurred. The liver showed a moderate amount of activity indicating drainage of silver by that organ.

The previous animal's litter mate, 51-72, showed quite a different picture than her sibling. The levels of radioactivity remaining are considerably higher than those in the previous dog. At autopsy the right lower lobe showed a grossly necrotic area of approximately 2½ cm. in diameter which largely resembled such areas found in the lung following the intrabronchial introduction of the gold colloid alone. The right lower lobe, that part which appeared more normal, contained over four times the concentration of radioactivity as the necrotic area in the right lower lobe (Table II). This may merely mean that the silver sloughed out following the dissipation of the irradiation due to the gold. In this animal the right mediastinal nodes, of which there were two, showed decidedly higher concentrations than the left. However, the left bronchial node had approximately twice the activity of the right bronchial node. Also there were two right tracheal nodes which showed a wide discrepancy in their concentration of radio silver and the left tracheal node contained a concentration equal to one of the right tracheals and four times the concentration of the other right tracheal node. The right axillary node taken as a control showed background activity as might be expected.

The third litter mate of this group, 51-73, like 51-71, showed no gross lesion of the right lower lobe into which the colloid had presumably been insufflated. All lung blocks had exceedingly low activities (Table III). The left and particularly right mediastinal node showed high levels of silver activity and the right tracheal node showed moderately high levels. Other nodes were quite low in their silver content. All nodes and lung tissues from this animal were counted on four occasions and the activities were essentially the same in all instances indicating that the latter were derived from the Ag^{110} and none from any residual gold activities.

TABLE II. 51-72. INTRABRONCHIAL Ag^{110} COATED Au^{198} (50 MC.) COLLOID

	WEIGHT OF NODE IN GM.	COUNTS PER MIN. PER GM.	COUNTS PER MIN. PER GLAND
L. mediastinal	0.035	90,000	3,146
R. mediastinal A	0.03	250,000	7,608
R. mediastinal B	0.025	360,000	9,064
L. bronchial	0.30	3,400	1,028
R. bronchial	0.14	1,940	272
L. tracheal	0.07	23,500	1,648
R. tracheal A	0.14	6,300	887
R. tracheal B	0.05	21,700	1,089
Bifurcation A	0.15	22,600	3,384
Bifurcation B	0.15	11,000	1,655
R. axillary	0.075	133	10
	WEIGHT OF TISSUE SAMPLE IN GM.	COUNTS PER MIN. PER GM.	
L. upper lung	0.21	53	
L. lower lung	0.58	15	
R. upper lung	0.53	430	
R. middle lung	0.27	1,380	
R. lower lung	0.68	14,500	
R. lower necrotic	0.64	2,900	
Liver	0.72	110	

Sacrificed after 42 days

Dog 52-183 was a mongrel about 2½ years old, weighing forty-five pounds, to whom was administered 120 mc. of a silver-coated gold colloid intrabronchially. After six days, during which period he appeared to be in excellent clinical condition, the animal was sacrificed with nembutal. The lungs on both the right and left sides were edematous and when cut exuded a free-running serous fluid. Otherwise there was nothing grossly of interest.

The radioactivity data are shown in Table IV where it is indicated that there was some spilling over of the colloid from one lobe of the right lung to

TABLE III. 51-73. INTRABRONCHIAL Ag^{110} COATED Au^{198} (50 MC.) COLLOID

DATE COUNTED		3/15/52	3/17/52	3/19/52	3/25/52	
	WEIGHT OF NODE IN GM.	COUNTS PER MIN. PER GM.	COUNTS PER MIN. PER GM.	COUNTS PER MIN. PER GM.	COUNTS PER MIN. PER GM.	COUNTS PER MIN. PER GLAND
L. mediastinal	0.05	66,000	70,000	67,000	63,000	3,300
R. mediastinal	0.07	174,000	167,000	197,000	180,000	12,200
L. bronchial	0.30	47	20	13	30	14
R. bronchial	0.17	106	47	41	47	18
L. tracheal	0.05	4,000	3,800	3,700	3,600	200
R. tracheal	0.03	21,200	18,800	19,000	18,000	639
Bifurcation A	0.22	41	46	32	18	9
Bifurcation B	0.69	51	26	32	41	35
R. axillary	2.28	4	8	2	4	9
	WEIGHT OF TISSUE SAMPLE IN GM.					
L. upper lung	0.67	50	31	24	13	
L. lower lung	0.91	19	11	8	7	
R. upper lung	0.58	33	36	29	22	
R. middle lung	0.70	17	20	7	—	
R. lower lung	0.71	25	35	6	21	

Sacrificed at end of 42 days

another. Of chief interest, however, is the fact that all of the nodes taken from the thorax showed values of radioactivity concentrations approximating that of the treated lobe or as high as eighteen times as much in the case of the right mediastinal node. As has been pointed out before, there is indication of excellent cross drainage as shown by the activities in the nodes taken from the left side. From a standpoint of time element this animal showed an excellent degree of lymphatic drainage, and adequate irradiation of all the thoracic lymph nodes was accomplished. One may only approximate the roentgen dosage received by the lymph nodes, assuming uniform distribution of the colloid throughout the node.

TABLE IV. 52-183. INTRABRONCHIAL Ag COATED Au^{198} COLLOID (120 MC.)

	WEIGHT OF NODE	COUNTS PER MIN. PER GM.	COUNTS PER MIN. PER GLAND	PER CENT TREATED LUNG
L. mediastinal	0.09	19,100	1,730	1,310
R. mediastinal	0.11	26,500	2,925	1,830
L. sternal	0.73	4,700	3,430	325
R. sternal	0.81	3,560	2,840	245
L. tracheal	0.13	7,280	947	500
R. tracheal	0.28	15,800	4,440	1,090
L. bronchial	0.36	1,260	452	87
R. bronchial	0.10	1,530	153	107
Bifurcation	0.16	5,050	810	350
L. upper lung		216		15
L. lower lung		64		4
R. upper lung		690		47
R. middle lung		1,450		100
R. lower lung		1,050		72
Liver		49		3.4
Heart		26		1.8

Sacrificed after 6 days

In order to calculate the equivalent roentgen dosage delivered to the various tissues, one is forced to make a number of assumptions. Among these is one, admittedly incorrect, to the effect that radioactive material remains in the same site at which it was found at time of sacrifice. From the various animals studied in this group this is obviously not true but for the purposes of an estimate we may take in animal 52-183 the distribution as found at the end of six days. In this animal we must further assume that the tissues examined contained the total of 120 mc. of gold administered. Taking an average lung lobe as 40 Gm. and the liver of this animal to be 450 Gm., and making the necessary calculations using the K_β for gold of 76, we find that the approximate beta equivalent roentgens delivered[3, 8] are approximately 2,500 e.r. to the liver. This is approximately that used in therapeutic management of chronic leukemias and is well tolerated by this organ.[5, 7] That amount of radiation delivered to the lung tissue will approximate 35,000 e. r. and to the lymph nodes approximately one-quarter of a million roentgens.

Dog 52-186, a male adult mongrel chow about 2 years old, had 50 mc. of a silver-coated gold colloid administered by bronchoscope and the animal was sacrificed ten days later by the administration of intravenous nembutal. The

clinical condition of the animal in the interim was excellent. Although most of the radioactivity was found in the right lower lobe of the lung nevertheless both other lobes of the right lung showed fairly considerable amounts of activity (Table V). In contradistinction to the other animals described receiving therapeutic doses there was rather poor drainage of the colloid by the thoracic lymphatics. Only the lymph node at the bifurcation of the trachea had accumulated any appreciable amount of radioactivity and this was only 66 per cent of the concentration per gram of fresh tissue as that found in the treated lobe of the lung.

TABLE V. 52-186. AG COATED Au^{198} (50 MC.) COLLOID INTRABRONCHIALLY

	WEIGHT OF NODE IN GM.	COUNTS PER MIN. PER GM.	COUNTS PER MIN. PER GLAND	PER CENT TREATED LOBE
L. sternal node	.31	96	30	0.6
R. tracheal node	.21	43	9	0.2
L. mediastinal node	.07	415	29	0.6
R. mediastinal node (fat)	.78	520	405	8
L. bronchial node	.47	280	131	3
R. bronchial A node	.34	2,030	692	14
R. bronchial B node	.16	6,460	1,036	21
Bifurcation node	.69	4,680	3,235	66
Axillary node	.55	13	7	0.1
	WEIGHT OF SAMPLE IN GM.			
L. upper lung	.18	326	59	1.2
L. lower lung	.33	140	46	0.9
R. upper lung	.51	1,900	968	20
R. middle lung	.74	2,600	1,932	40
R. lower lung	.96	5,100	4,900	100
Liver	1.1	8	9	0.2

Sacrificed after 10 days

Dog 52-173, a male mongrel of about 2 years of age and weighing thirty-three pounds, was given a silver-coated gold colloid representing 85 mc. of Au^{198} into the right lower lung by bronchoscope. At the end of twenty-six days, during which time the clinical condition of the animal appeared to be excellent, he was sacrificed with nembutal. Approximately one liter of fluid was found in the pleural cavity although the lungs grossly appeared to be normal pink in color and there was no sign of hemorrhage. Activity measurements shown in Table VI indicate that drainage from the treated lung by this length of time was nearly complete. Distribution of radioactivity in the various thoracic lymph nodes was quite satisfactory reaching as much as twenty-four times that of the treated lung in the case of the left sternal node. It is again of interest that the left mediastinal, left bronchial, left sternal, and left tracheal nodes all showed higher activities than their corresponding right-sided nodes. Therefore there seemed to be a preferential drainage to the left side in this particular animal.

No attempt is made in this brief presentation to include a complete study of the pathology and histopathologic findings of the tissues and neither is there an attempt made to correlate comprehensively such findings with the radiation delivered. This will be done in more detail at a latter date. However, a number

TABLE VI. 52-173. AG COATED Au^{198} COLLOID INTRABRONCHIALLY (85 MC.)

	WEIGHT OF NODE IN GM.	COUNTS PER MIN. PER GM.	COUNTS PER MIN. PER NODE	PER CENT TREATED LUNG
L. mediastinal	.31	7,900	2,453	1,600
R. mediastinal	.14	8,100	1,131	740
L. bronchial	.24	750	180	118
R. bronchial	.20	835	167	108
L. sternal	.60	6,130	3,680	2,400
R. sternal	.77	4,050	3,117	2,000
L. tracheal	.37	930	343	220
R. tracheal	.80	81	65	43
L. bifurcation	.66	650	429	280
	WEIGHT OF SAMPLE IN GM.			
L. upper lung	.41	37		10
L. lower lung	.50	24		8
R. upper lung	.41	71		19
R. middle lung	.52	40		13
R. lower lung	.50	310		100
Liver	.79	530		270

Sacrificed 26 days following instillation

of selected sections varying widely in their residual radioactivity content were submitted for histopathologic examination and the results are briefly described here. It is to be noted that in general the changes seem to parallel quite closely the degree of residual activity present in the tissues. There are one or two exceptions. It should be pointed out that the columnated figures of radioactivity in Tables I to VI expressed in counts per minute per gram of fresh tissue are relative values, inasmuch as some samples were of such high activity, it was necessary to use adsorbers interposed between the mica window counter and the sample. In other instances the counts were relatively low due to protracted time intervals between insufflation of colloid and sacrifice and it was possible to count directly under the window.

Histopathology.—*

A. Dog 51-71

1. Left mediastinal lymph node was hyalinized with obliteration of afferent and efferent lymphatic channels and showed early calcific deposits. There were no marginal or medullary sinuses and no lymphatic or reticuloendothelial tissue present.

2. Right mediastinal lymph node was similar to left mediastinal lymph node with glassy hyalinization. There was fat necrosis with sheaths of fatty acid crystals and thrombosis of lymphatics.

3. Right bronchial lymph node showed hyperplastic lymphatic follicle. No radiation effects were evident. Marginal sinuses were difficult to make out.

4. *Right tracheal lymph: subtotal (80% ±) hyalinization with some* persistence of reticular elements.

5. Bifurcation was similar to right bronchial node.

*We are deeply indebted to Dr. R. L. Holman for his kindness in reviewing some of the histopathologic sections on these animals.

6. Lung: right lower lobe showed patchy fibrosis with a question of lipoidal type pneumonia. There was a questionable thrombosis of one of the blood vessels.

7. Lung: right lower lobe similar to but not as marked as in No. 6.

8. Left lower lobe showed patchy interstitial pneumonia with questionable foci of necrotizing arteritis.

Summary: (1) Lymph nodes can be obliterated by the technique employed. (2) In process of obliteration both lymphatic and reticuloendothelial tissue undergoes coagulative necrosis with subsequent hyalinization and calcific deposits. (3) Both afferent and efferent lymphatic channels appear to be obliterated in the process. (4) Reaction in the lung is not striking and is patchy.

B. Dog 51-72

1. Right mediastinal lymph node (a) showed complete hyalinization with collapsed sinusoids and a question of arteritis. Pigment in this node was felt probably to be anthracotic but the possibility of its representing silver or gold has not been excluded.

2. Right mediastinal lymph node (b) was similar to (a) in No. 1 and with arteritis.

3. Right bronchial lymph node presented a picture of atrophy of lymphatic tissue, reticuloendothelial hyperplasia, early necrosis and hemorrhage (especially about blood vessels). The architecture of the lymph node persisted in focal areas, both cortical and medullary.

4. Bifurcation node (a) was similar to but less marked than right bronchial lymph node No. 3. Some germinal centers were present.

5. Right axillary lymph node was similar to bifurcation No. 4.

6. Lung: left upper lobe showed minimal interstitial reaction with lipophages.

7. Lung: right lower lobe, had pneumonic process especially hemorrhage and slightly more marked than in No. 6.

8. Lung: right lower lobe, had large areas of coagulation necrosis with questionable thrombosis.

Summary: Findings in this dog were similar to those in 51-71 but in addition suggest: (1) The necrosis of the right lower lobe may be related to infarction. (2) Slight to moderate doses of radioactivity exposure lead to atrophy of lymphatic tissue with reticuloendothelial hyperplasia.

C. Dog 51-186

1. Right bronchial lymph node (a) showed early necrosis especially of lymphatic tissue as well as some reticuloendothelial hyperplasia. Marked anthracosis was noted especially in medullary part.

2. Right bronchial lymph node was similar to (a) just described.

3. Right tracheal lymph node had few active germinal centers and showed early reticuloendothelial hyperplasia. There was erythrophagocytosis in medullary sinuses.

4. Bifurcation lymph node was similar to No. 3 with large avid phagocytes engulfing necrotic lymphocytes in remnants of germinal centers.

5. Left sternal lymph node was similar to bifurcation No. 4.
6. Axillary lymph node was similar to No. 4 and No. 5.
7. Liver was not noteworthy.

Summary: In ten days there was stimulation, exhaustion, and phagocytosis in germinal areas, and some reticuloendothelial hyperplasia with hemorrhage in sinusoids. Apparently this represents the early stages in the process that ends in hyalinization.

DISCUSSION

There are several important points which must be considered in light of the purpose of the described experiments. If we hew to the proposed line of attack of bronchiogenic tumor by the instillation of these radioactive colloids intrabronchially prior to pneumonectomy we are presupposing that such colloidal material would behave in the diseased human organism similarly to that which is found to obtain in the normal dog's pulmonary system.

Several points of interest are worthy of mention. The frequency with which the contralateral nodes concentrated radioactivity is eloquent evidence of cross drainage of the lymphatics and fits in with the findings of metastatic processes in breast and lung tumors. However, drainage by the various nodes appears to be quite haphazard. For example, in Dog 51-71 there were two bronchial nodes found on the left side; one of which showed 250 times the concentration of radioactivity as the other. Again in Dog 51-72 two right tracheal nodes differed in concentration of the silver by a factor of 3. In 52-183 the distribution of the coated colloid was excellent regarding the findings in the thoracic lymph nodes. On the other hand in certain other animals there was a disappointing amount of concentration of the colloid in some lymph nodes and inadequate irradiation of such tissue resulted.

We note that in Dog 51-72 the lungs showed, especially in the lobe into which the material was administered, considerably higher radioactivities than in the two dogs, 51-71 and 51-73, the latter having manifested no gross pathologic lesions. The amount of material at hand is too meager to enable us to speculate why in this particular animal the silver was not drained as completely as in the others. In the case of Dog 51-73, repeated measurements were made of the lymph nodes and lung blocks in order to reassure ourselves that the residual activities were due to the Ag^{110} isotope and the data substantiate this (Table III). Again, in this animal the tremendous variations in the radioactivity concentrations in the various nodes are apparent. In several other animals where sacrifice occurred at rather belated intervals the concentration in the lymph nodes was many times that in the insufflated lobe of the lung. Such intervals between instillation and sacrifice sometimes are of a length which is far longer than the time necessary for the dissipation of all the radiation from the gold nucleus. Therefore it may well be that for the most successful irradiation of such nodes, we shall ultimately be well advised to resort to use of the Ag^{111} isotope which, having a half life of seven and one-half days, may be much more suitable for prolonged irradiation in metastatic bronchiogenic tumor.

As to the uniformity of distribution of the colloid in the lymph nodes it might be said that the few autoradiographs which have been made of these tissues suggest a rather spotty distribution. A great deal of further experimentation must be carried out to assess the factors which determine the deposition of colloid in different lymph nodes.

The extremely variable distribution of the radioactive material in tissues demands a large margin of safety, regarding both minimal dosage compatible with a cancericidal effect in the lymphatic system and a dosage which is not too deleterious to other tissues. If, for instance, one considers Dog 52-173 (Table IV) which was sacrificed twenty-six days following intrabronchial instillation of the colloid it would be found that the liver radiation dosage was quite considerable. However, we have in other studies found that this organ is capable of withstanding relatively massive amounts of irradiation before irreversible pathologic changes are undergone.[5, 7] The wide variations in the dosage of radiation delivered to the lymph nodes is partially compensated for by the fact that we are, in most cases, dealing with many times the probable cancericidal dose.

Taking into consideration the fact that the half-path of the gold beta particles is 0.4 mm. in tissue this would allow for the order of magnitude of only forty cell diameters for a lethal effect to be attained regarding malignant cells. On the other hand, in working in the order of 10^5 equivalent beta roentgens, we are in a range where there is probably no inconsiderable amount of effect due to the relatively small gamma component of this isotope and in the final analysis it may well be that a satisfactory therapeutic dose may be realized. Of course, as stated elsewhere, the foregoing values are based on normal dog tissue and it remains to be seen whether such factors as blockage of lymphatic channels by tumor tissue in the cases of human malignancy will operate to render impossible the delivery of adequate amounts of irradiation under such conditions.

Throughout all such conjecture one must keep in mind that all calculations at the present time are carried out on a basis of rather sweeping assumptions due to the transient behavior of the radioactive colloid in the tissues under consideration. It is not likely that dosage calculations shall ever be as simple as those for x-irradiation or for implants of long-lived material (for example, radium) and it is well recognized that the difficulty of calculating irradiation dosage for these latter is complicated enough.

It should be noted in favor of the likelihood of obtaining adequate irradiation despite inhomogeneous distribution of the colloid in the nodes that, for example, in Dog 51-71, several of the nodes which had received the largest amount of radiation had atrophied to the point that they weighed only from 40 to 90 mg. The total dimensions of the nodes at such a time were such that adequate beta irradiation must have been accomplished in spite of the short path of the beta particle of the isotope used. In the illustration of the photomicrograph of such a node (Fig. 1), the entire node is represented. Of course we cannot say for certain how much of the atrophy preceded the partial or complete dissipation of the energy from the contained isotope.

What appears at present to be a limiting factor in the efficacy of the instillation of radioactive colloids intrabronchially as a form of treatment is represented by the widely discrepant activities exhibited by the various lymph nodes studied. Our knowledge of the lymphatic drainage of the thoracic cavity as well as other areas of the body is in a parlous state. Further use of colloids with and without concomitant studies of the effect of localized inflammatory processes may assist in divulging which factors are operating to cause concentration of these materials in certain nodes. The dog is notoriously prone to develop pulmonary infections and it may well be that certain nodes at various times are subject to inflammatory reaction and therefore may concentrate materials to a different extent than near-by nodes.

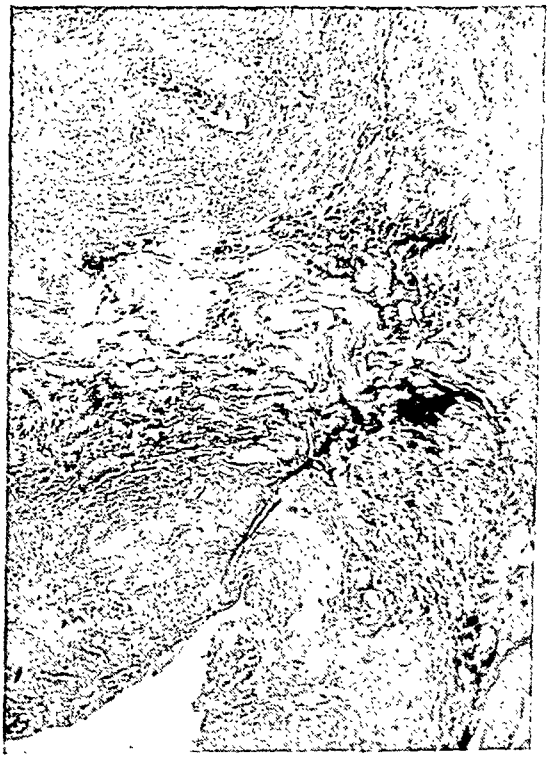


Fig. 1.—Dog 51-72. Right mediastinal node. (Hematoxylin and eosin; ×150.)

As stated before in dealing with human pathologic conditions the probability of lymphatics being plugged by tumor tissue and, therefore, being impervious to transport of the colloid to the nodes must be considered. Studies are underway in several far-advanced cases of bronchiogenic tumor in human beings to determine the degree of deposition of such colloids under these circumstances. The possibility should be kept in mind that development of collateral lymphatic drainage under such circumstances may possibly come to the aid of the therapist.

SUMMARY

The distribution of silver-coated radioactive colloidal gold particles, following their intrabronchial administration in dogs, has been studied at levels of radioactivity which are estimated to be in a therapeutic range for treatment of metastatic lesions in bronchiogenic tumors.

A short series of experiments in which the silver coat as well as the gold nucleus were radioactive indicates that the silver coat is gradually leached off in the lung and is carried to the liver for ultimate secretion, probably by the biliary tract.

It is found that the silver assists in the transport of the radioactive gold nucleus by way of the lymphatics to the thoracic nodes thus facilitating irradiation of these common sites of metastatic processes.

Estimated roentgen equivalent dosages to various tissues of interest would suggest that the amount of irradiation encountered by the liver is well within tolerable limits by a factor of perhaps 10 times. Dosages to the lung into which the material was introduced are relatively high but this tissue is considered expendable since in most cases pneumonectomy is planned when treatment is anticipated. Dosages to the lymph nodes are estimated to be of the order of several hundred thousands equivalent beta roentgens. It is felt that discrepancies in uniformity of distribution in such tissues would be well taken care of by the large factor of safety over estimated cancericidal doses necessary to deal with metastatic processes. The possibility of obstruction in the cases of metastatic tumor involving lymphatic channels is considered. Also the possibility of collateral lymphatic drainage under such conditions is hypothecated.

Study of drainage by the lymph nodes in cases of bronchiogenic tumor in human beings is obviously necessary before the distribution of radiation can be depended upon as a therapeutic procedure.

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INTRAPERICARDIAL DISSECTION IN LEFT PNEUMONECTOMY FOR BRONCHIOGENIC CARCINOMA

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PULMONARY resection is generally accepted as the treatment of choice in cases of carcinoma of the lung in which the lesion is operable. There is as yet, however, no general agreement as to what constitutes an adequate radical resection in cases of cancer of the lung. One approach to a more extensive resection of tissue has been the employment of intrapericardial dissection.

Intrapericardial dissection is practicable in the performance of both right and left pneumonectomy. The problem is somewhat different in cases in which a right pneumonectomy is necessary as contrasted with those in which a left pneumonectomy is necessary owing to the anatomic differences pertaining in the two hemithoraces. To avoid confusion regarding this, we shall limit this presentation to a discussion of certain features of left pneumonectomy concerned with the intrapericardial handling of the vessels in this operation.

HISTORICAL BACKGROUND

The first successful pneumonectomies were performed by Nissen,[1] Haight,[2] and Graham and Singer[3] employing the tourniquet technique; however, it was Rienhoff[4] who early advocated the individual dissection, ligation, and division of the hilar structures, and perfected the technique[5] used by most surgeons today in performing the standard pneumonectomy.

Consistent with the general principles of cancer surgery, en bloc dissection of the tumor and surrounding involved structures should be effected in operations for cancer of the lung. Credit for extending the limits of dissection in pneumonectomy to include the intrapericardial portion of the vessels and mediastinal nodes goes to Allison.[6] He first described the technique in 1946, stressing the fact that tissues should be removed in continuity. Later, in discussing a paper by Churchill and co-workers,[7] he stated that "to do either a lobectomy or a pneumonectomy and then pick out isolated glands from the mediastinum is to forget our surgical pathology." While in 1942 his extrapericardial dissections outnumbered the intrapericardial ones 6 to 1, in 1949, 23 out of 27 pneumonectomies were radical resections in which a portion of the pericardium was excised. Of interest also was that during this period, his operative mortality rate dropped from 23 per cent to 4 per cent.

In this country, Gibbon and co-workers[8] were the first to stress the merits of intrapericardial ligation and division of the vessels when dealing with bronchiogenic carcinoma. In 10 out of 20 reported pneumonectomies,[8] such a procedure was essential if the lesion was to be resected at all. In a later communication, Healey and Gibbon[9] reported 44 out of 102 pneumonectomies

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performed by this technique and discussed their findings on intrapericardial anatomy based on dissections of 185 adult cadavers. Other recent reports[10, 11] have mentioned the usefulness of intrapericardial dissection.

INDICATIONS

There are two instances in which it seems mandatory that the surgeon enter the pericardial sac for the securing of the pulmonary vessels if he is to effect left pneumonectomy. The first exists in cases in which the carcinoma has extended in such a fashion as to render extremely hazardous or impossible the securing of the pulmonary artery or of the pulmonary veins outside the pericardium in the usual fashion. The second situation pertains in cases of carcinoma of the lung in which the involvement has progressed so that there are nodes and tissues involved overlying the pulmonary artery or perhaps along the inferior pulmonary vein or along the mediastinum medial to the left main bronchus and behind the heart. The tissues at these sites cannot be adequately excised en bloc by the standard left pneumonectomy. However, it has been our experience that these tissues can be radically and accurately excised along with the lung by securing the pulmonary vessels within the pericardium, and by excising a portion of the pericardium along with the tissues overlying the aortic arch and pulmonary artery and those lying behind the pericardium.

There remain many cases of carcinoma of the lung in which it is technically feasible to remove the lung without entering the pericardium. Nonetheless, by entering it, a more extensive removal of tissue is possible. There is not yet sufficient evidence to permit a conclusion as to whether such an approach is advisable in all operations for bronchiogenic carcinoma. At present, it is our belief that in peripheral carcinoma of the lung without nodal involvement, lobectomy is indicated. If the lesion is centrally located, but small and without local extension or significant involvement of lymph nodes, pneumonectomy without intrapericardial dissection is probably advisable. However, if there is any degree of local invasion or if there is considerable nodal involvement, a more radical pneumonectomy such as will be described is believed by us to be advisable. We recognize that such an extended operation may become advisable for all centrally located carcinomas of the lung when further data regarding mortality and curability become available.

TECHNIQUE

For intrapericardial dissection of the pulmonary vessels on the left side, the pericardium is incised over the region of the left atrial appendage; this incision often is made just posterior to the left phrenic nerve. If there is considerable invasion of the pericardium, the incision is made anterior to the phrenic nerve to allow a more generous excision of it (Fig. 1). The incision is carried cephalad to such a point that the pulmonary artery and aorta are widely exposed. From the lower angle of the pericardial incision and at a right angle to it, an incision is made posteriorly, and wide exposure of the pulmonary artery and superior and inferior pulmonary veins is obtained. The

superior pulmonary vein is dissected out and doubly ligated flush with the left atrial wall and divided. The inferior pulmonary vein is similarly treated, again ligating it flush with the left atrial wall.

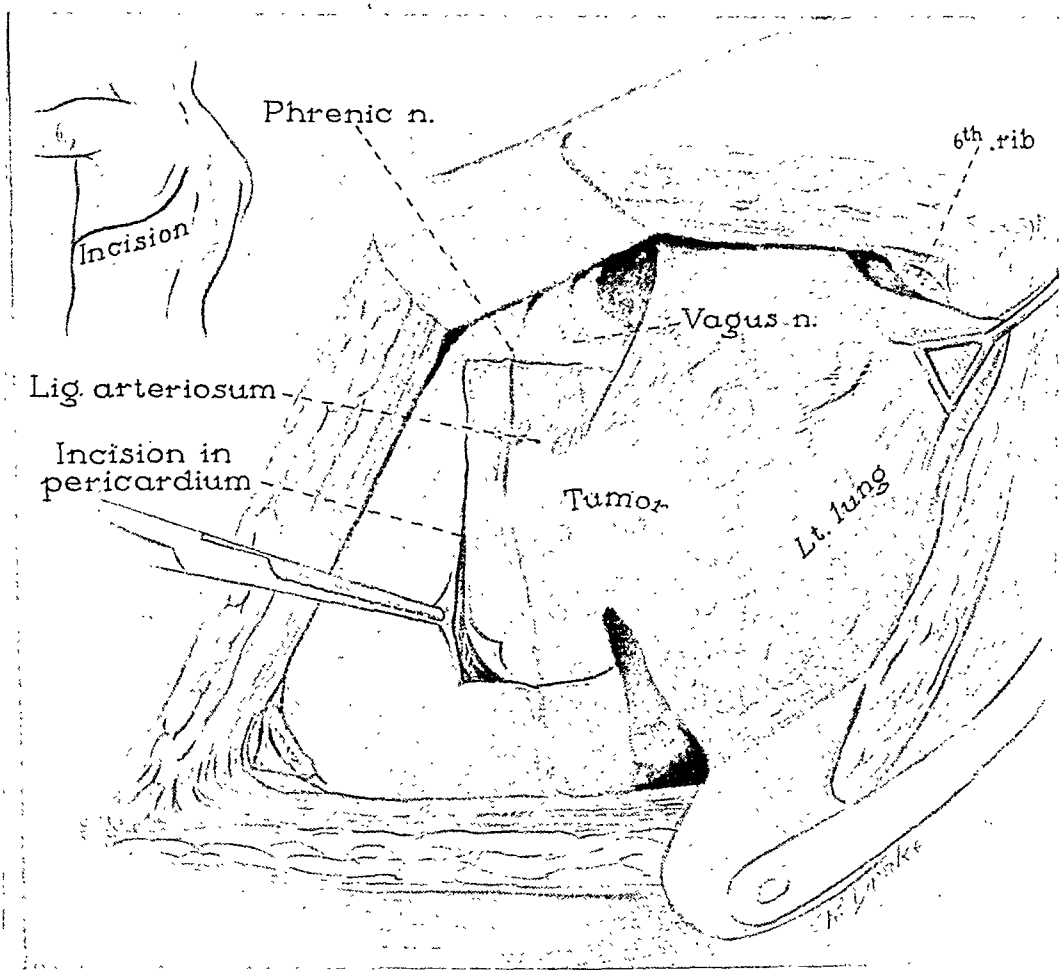


Fig. 1.—Intrapericardial dissection. Initial incision in the pericardium and mediastinal pleura. On occasions when the tumor does not invade tissue near the phrenic nerve, the incision may be made just posterior to the phrenic nerve in order to preserve it.

Next, the left pulmonary artery is dissected out and completely mobilized up to the ligamentum arteriosum. The artery is then doubly or triply ligated just distal to the ligamentum arteriosum and divided. During the mobilization of the pulmonary artery, it is important to sweep all the overlying pericardium and soft tissues including the lymph nodes back toward the lung so as to obtain a wide resection of them (Fig. 2).

The dissection is then carried cephalad over the aortic arch, sweeping all the soft tissues and lymph nodes in this region down with the resected specimen. It is usually necessary in this maneuver to sacrifice the vagus nerve. Usually, but not always, this can be done distal to the origin of the recurrent

laryngeal nerve. The soft tissues in the angle between the bronchus posteriorly and the pulmonary artery anteriorly are all included in the resection.

Once the pulmonary artery has been divided and a portion of the pericardium at this site swept back toward the lung, it becomes possible to include with the resection the peribronchial lymph nodes and subcorynal nodes. Posteriorly and inferiorly, the lymph nodes along the bronchus are likewise swept down to be included along with the resected specimen (Fig. 3). The bronchus is secured about 4 mm. from the coryna and is closed with interrupted silk sutures after the method of Sweet.[12] By careful and accurate dissection, the lung is then peeled away from the mediastinum, great pains being taken to include a wide excision of the soft tissues overlying the aorta and esophagus which include, of course, a number of mediastinal lymph nodes.

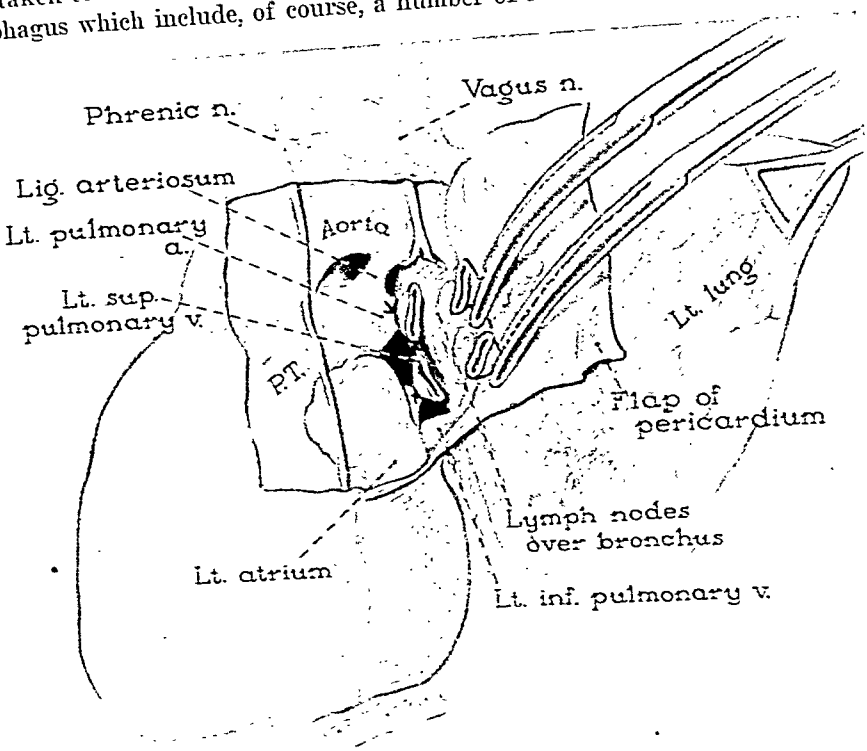


Fig. 2.—Further stage in intrapericardial dissection. In practice, the phrenic nerve is resected along with the pericardial flap in a case such as this, but in order to clarify relations it has been left in situ in this figure and in Fig. 3.

At the end of the resection, there is a defect in the pericardium that sometimes measures 6 to 7 cm. in diameter. We have as yet seen no ill effect from leaving this defect. A flap of pleura is turned down to cover the bronchial stump. The thoracic wall is closed in layers. Drainage is not employed.

COMMENT

There exists at the present time a wide disparity in the views held by surgeons concerning the extirpative treatment of malignant lesions. There are those surgeons who feel that extended operations for carcinomas of bor-

derline operability are not wise and that very few if any patients will be benefited permanently by such procedures. There are others who feel that radical extensive operations should be done not only for carcinomas of borderline operability but for all carcinomas. There are excellent arguments for both schools of thought and it is impossible at this time to state dogmatically that one or the other is correct.

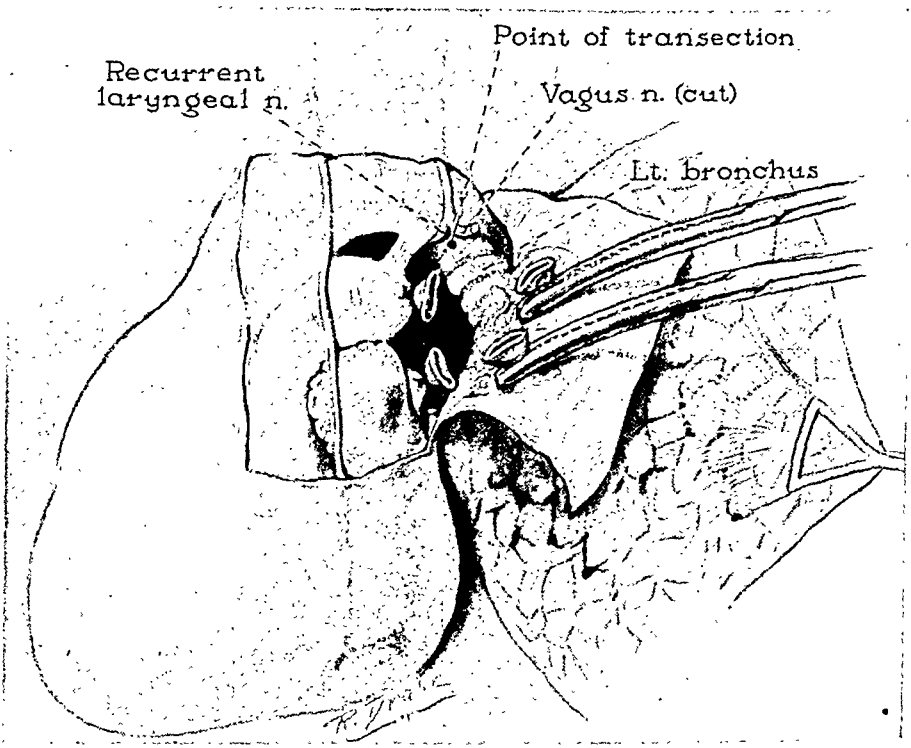


Fig. 3.—Still later stage in intrapericardial dissection. Note that the nodes and soft tissues have been swept down from the upper reaches of the left main bronchus, in preparation for transection of this bronchus just distal to the coryna.

In most cases of centrally located carcinoma of the lung, it has seemed to us advisable to carry out a resection which is somewhat more extensive than the standard pneumonectomy. On the left side, this implies not only ligation of the pulmonary vessels within the pericardium but also wide removal of a portion of the pericardium and all the lymph nodes surrounding the extrapericardial portions of these vessels. It implies also wide removal of the soft tissues beneath the aortic arch and all the lymph nodes going up along the left main bronchus to the coryna. It implies high division of the left main bronchus so as to get well above the carcinoma.

It is our opinion that such an extended pneumonectomy should offer most patients requiring the operation some better hope of cure than the traditional operation in which admittedly many mediastinal lymph nodes that could be removed by the more extended procedure are left behind. Furthermore, there

is little question that certain lesions which would be inoperable because of the involvement around the pulmonary vessels become, in truth, operable if one is willing to proceed with intrapericardial dissection.

The extended operation has been carried out by many individuals as well as by ourselves without a significant increase in the risk of the operation. Our experience is relatively small; we have performed intrapericardial dissection on the left side in only 18 cases. In this group, there have been no complications of any kind, save a single fatal one. In this case, the operation proceeded satisfactorily and the patient's condition was good for about two hours after the operation. Massive hemorrhage then occurred into the left side of the thorax, and at re-exploration we found the ligatures had become dislodged from the central end of the pulmonary artery. Bleeding was controlled but the patient suffered such a prolonged period of cerebral anoxia that he succumbed within twenty-four hours. This complication was probably the result of a technical error, the occurrence of which can be minimized by extreme care in the handling of the vessels.

SUMMARY

The advantages and technique of radical left pneumonectomy employing intrapericardial dissection of the vessels are presented. It is emphasized that by this technique a more radical resection of possible cancer-bearing tissue is effected. Furthermore, certain lesions which would be inoperable because of involvement around the pulmonary vessels become operable when intrapericardial dissection is employed.

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RESPIRATORY ACIDOSIS DURING INTRATHORACIC SURGERY: THE (OVERHOLT) PRONE POSITION

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RESPIRATORY acidosis as a complication of thoracic surgery was described as early as 1938 by Crafoord.[3] Quantitative investigations in man by several observers[1, 6, 8] have confirmed this earlier finding that a high arterial blood carbon dioxide tension frequently occurs during intrathoracic pulmonary resection, but they are not in agreement as to the mechanisms that impede the excretion of carbon dioxide. Taylor and Roos[13] report that the diminished alveolar ventilation is caused by the depressant effect of ether anesthesia on the respiratory center. Gibbon and associates[6] state that the acidosis is a result of inadequate pulmonary ventilation, caused by an open pneumothorax and not by the lateral position. Beecher and his associates[2] report that the accumulation of carbon dioxide in the blood during surgery of the open thorax is related to the position of the patient, and that the prone (Overholt) and supine positions are the most favorable for the excretion of carbon dioxide (unassisted respiration).

It seemed that further studies might elucidate the causes of respiratory acidosis during thoracic surgery. If, as Beecher states, the prone position is the most favorable for the excretion of carbon dioxide during intrathoracic surgery, analysis of the variables that may disturb the acid-base balance during anesthesia and surgery would help clarify the mechanisms involved in producing high CO_2 tensions in the blood. In order to evaluate fully the effect of the Overholt prone position on carbon dioxide excretion during pulmonary surgery, such variables as preoperative sedation, depth of ether anesthesia, change of position, opening of the pleural cavity, and pulmonary resection have been serially analyzed in this study.

Factual data are presented to show that high carbon dioxide tension occurs in the prone position, with respiration neither assisted nor controlled. The open thorax and the depth of ether anesthesia are the two most important factors in producing a respiratory acidosis during pulmonary surgery.

PREOPERATIVE SEDATION, ANESTHETIC TECHNIQUES, AND AGENTS

Sedation.—All patients in this series received 0.1 Gm. pentobarbital sodium, subcutaneously, two hours before induction of anesthesia, and one hour later received morphine sulfate (or Demerol) plus scopolamine. The selection and

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dosage of morphine or Demerol varied according to the patient's age, sex, and physical status. The dose of scopolamine was 0.6 mg., morphine ranged from 6 to 10 mg., and Demerol from 25 to 100 mg.

Anesthesia.—Ether-oxygen was the maintenance anesthetic agent. The patients were divided into two groups, according to the depth of anesthesia: Group I, light-ether anesthesia; Group II, moderate-depth anesthesia. In Group I endotracheal intubation was performed under topical laryngotracheal 5 per cent cocaine anesthesia. General anesthesia was induced with a 2.5 per cent intravenous solution of Pentothal sodium after the patient was placed in the prone position. Anesthesia was maintained in a very light plane with ether and oxygen. Eyelid reflexes and eyeball movement were active throughout the procedure. The patient periodically reacted to painful stimuli by movement of legs or arms. In Group II, while the patient was in the supine position, general anesthesia was induced with nitrous oxide and oxygen and maintained with ether-oxygen. The muscular relaxation obtained permitted endotracheal intubation and the patient was placed in the prone position. A moderate plane of the surgical stage of ether anesthesia was maintained throughout the operative procedure.

Anesthesia Technique.—An imbedded Latex endotracheal tube with inflatable cuff was connected in series with a to-and-fro Waters' canister and bag. The endotracheal balloon was inflated to obtain an airtight fit. The to-and-fro canister was filled with fresh Baralyme in each case and changed every hour. Respirations were spontaneous throughout the entire anesthetic and surgical procedure and the patient's respiratory exchange was neither assisted nor controlled at any time.

The depth of anesthesia of the patients in Group I is identified in Table I by the letter "L" and in Group II by the letter "D."

Position.—The patient was placed face-down on an Overholt-Compere table,[3] positioned so that the crest of the ilium was supported by the edge of the operating table and the chest by an adjustable pad placed at the cephalad third of the sternum. This allowed the chest cage to expand freely. The head was supported by a modified cerebellar headrest.

General Methods.—The acid-base equilibrium was studied in twenty-three patients. Twenty-two patients were anesthetized with ether; one patient had a pneumonectomy under paravertebral procaine 1 per cent block with endotracheal oxygen. Ninety-six serial acid-base analyses were obtained.

Arterial blood samples were drawn in the following sequence: (1) before the administration of the preoperative sedation, (2) sixty minutes after the administration of the preanesthetic sedation, (3) after the patient was placed in the prone position and anesthetized, (4) before the pleura was opened, (5) after the pleura was opened, before the lobe or lung had been removed, (6) after lobectomy or pneumonectomy with the pleura still open, and (7) at the conclusion of the operation.

Respiratory studies of the tidal exchange, respiratory rate, and respiratory minute volume were obtained by connecting a balanced-type Benedict-Roth spirometer directly to the endotracheal tube. Measurements were made with the patient in the supine and prone positions during the operative procedure.

One medical staff anesthetist administered the anesthesia to all patients studied during this investigation. All patients were operated on by one surgeon.*

Chemical Methods.—Blood was drawn from the femoral artery before the administration of the preanesthetic medication. A Cournand needle was inserted into the radial artery and taped to the wrist with adhesive plaster strips before induction of anesthesia. Arterial blood for oxygen and carbon dioxide determinations was collected by slightly heparinized syringes and transferred to oxalated tonometers over mercury. Arterial blood for pH was collected under oil and centrifuged. The pH of the serum was determined by a Beckman model G glass electrode. This instrument was standardized before and after each determination with a standard buffer solution having a pH of 7.00. The pH was corrected for a temperature of 38° C.[4]

The arterial oxygen content, oxygen capacity, and carbon dioxide content determinations were made on 1 c.c. samples of whole blood by the method of Van Slyke and Neill. The Goldstein modification of the Van Slyke method was used when ether was present in the blood.[7] Carbon dioxide tension was calculated from the carbon dioxide content in volumes per cent, and pH value from the nomogram of Peters and Van Slyke.[10] Values higher than could be read from the nomogram were calculated by the Henderson-Hasselbalch equation. Total CO_2 values in millimols per liter were read from the chart of Peters and Van Slyke.[11] The bicarbonate value of the plasma $[(HCO_3^-)_p]$ was calculated. The calculated data depend mainly on accurate determinations of the pH. In order to obtain accurate determinations within the narrowest margin of error, the analyses of blood gases and pH were all performed by one individual expertly trained in these techniques.

RESULTS

The composite data of the investigation are given in Table I.

The effect of preoperative sedation on the arterial blood carbon dioxide and oxygen saturation is listed in Tables II and III. Analysis of these tables indicates that preoperative sedation in some instances may produce retention of carbon dioxide (see Table II). From Table III it can be seen that moderate doses of pentobarbital with morphine and scopolamine do not cause respiratory acidosis in most patients. Statistical analysis of Table II shows that the T test for examination of the significance in differences in pCO_2 before and after sedation gives the value $T = 4.44$, with 7 degrees of freedom. This value is significant at the .01 significance level. In Table III the observation of the effect of sedation in eighteen patients reveals a correlation coefficient between "time after sedation" and pCO_2 of $-.27$. This figure indicates that

*Dr. Francis Woods.

TABLE I. ACID-BASE EQUILIBRIUM, THORACIC ANESTHESIA, (OVERHOLT) PRONE POSITION, SPONTANEOUS RESPIRATION

CASE	AGE	SEX	MEDICATION: MORPH. (MG.)	MEDICATION: SCOP. (MG.)	DIAGNOSIS AND OPERATION	ANESTHESIA: CONDITIONS	ANESTHESIA: TIME (MIN.)	ANESTHESIA: DEPTH	POSITION	(MIN.)	PLEURA OPEN (MIN.)	pH, 38° C.	pCO_2 (MM. HG)	TOTAL CO_2 (mM./L)	(HCO_3^-) (mM./L)	HGB. (GM./100)	β	(HCO_3^-) CORRECTED TO pH 7.4 (mM./L)	CHANGE IN FIXED ACID	BLOOD O_2 SAT. (%)
1	28	F	8.1	0.324	A-V aneurysm	Control			Supine			7.45	28	19.8	18.9	14.7	28.8	20.4		89
						Post-sed.	60		Supine			7.19	50	19.5	18.0	14.7	28.8	11.9	+ 8.5	89
					Lobectomy	Ether/Oxygen	105	D	Prone	110	35	7.00	82	20.6	18.1	14.7	28.8	6.6	+13.8	81
2	28	F	11.0	0.432	Tuberculosis	Control			Supine			7.42	34	22.0	21.0	10.1	22.4	21.2		97
						Post-sed.	60		Supine			7.21	52	21.4	19.8	10.7	23.3	15.4	+ 5.8	95
					Lobectomy	Ether/Oxygen	40	L	Prone	65		7.43	30	20.0	19.1	10.7	23.3	19.8	+ 1.4	98
3	48	F	11.0	0.432	Bronchiectasis	Control			Supine			7.43	41	27.1	25.9	14.1	27.9	26.7		92
						Post-sed.	87		Supine			7.31	50	24.7	23.2	13.9	27.7	20.7	+ 6.0	87
					Pneumonectomy	Ether/Oxygen	10		Supine			7.23	64	26.6	24.7	14.0	27.9	20.0	+ 6.7	90
						Ether/Oxygen	70		Prone	45		7.30	56	27.0	25.3	14.0	27.9	22.5	+ 4.2	92
						Ether/Oxygen	132	D	Prone	107	52	7.08	109	34.1	30.8	14.0	27.9	21.9	+ 4.8	79
4	48	M	11.0	0.432	Lipoma	Control						7.39	43	25.6	24.3	14.7	28.8	24.0		99
						Post-sed.	45					7.31	54	27.0	25.4	14.7	28.8	22.8	+ 1.2	96
					Thoracotomy	Ether/Oxygen	15		Supine			7.33	54	28.4	26.8	14.7	28.8	24.8	− 0.8	100
						Ether/Oxygen	60	L	Prone	40	5	7.30	43	24.2	22.9	14.7	28.8	21.8	+ 2.2	100
5	50	M	11.0	0.432	Carcinoma	Control			Supine			7.30	47	22.9	21.5	11.3	24.1	19.1		90
						Post-sed.	60		Supine			7.21	52	21.3	19.7	11.8	24.9	15.0	+ 4.1	93
					Pneumonectomy	Post-sed.	95		Supine			7.10	31	19.6	18.7	11.8	24.9	18.7	+ 0.4	98
						Post-sed.	110		Prone	15		7.37	34	20.0	19.0	11.8	24.9	18.2	+ 0.9	98
						Ether/Oxygen	60		Prone	105	15	7.03	83	22.0	19.5	13.3	26.9	9.6	+ 9.5	89
						Ether/Oxygen	150	D	Prone	195	105	7.14	72	24.3	22.1	13.3	26.9	15.1	+ 4.0	90
6	56	F	8.1	0.324	Carcinoma	Control						7.35	42	23.5	22.2	11.7	24.7	21.0		89
						Post-sed.	75					7.31	44	22.3	21.0	11.7	24.7	18.8	+ 2.2	93
					Pneumonectomy	Ether/Oxygen	35		Supine			7.25	54	23.8	22.2	11.7	24.7	18.5	+ 2.5	97
						Ether/Oxygen	85		Prone	45		7.35	37	20.6	19.5	11.6	24.5	18.3	+ 2.7	93
						Ether/Oxygen	130	D	Prone	90	40	7.10	76	23.6	21.3	12.3	25.5	13.7	+ 7.3	96
7	63	M	75.0 (Dem.)	0.324	Carcinoma	Control						7.41	45	28.5	27.1	13.9	27.7	27.4		92
						Post-sed.	65		Supine			7.21	66	25.9	23.9	13.9	27.7	18.7	+ 8.7	90
					Pneumonectomy	Ether/Oxygen	90		Supine			7.23	69	28.5	26.4	13.9	27.7	21.7	+ 5.7	100
						Ether/Oxygen	140		Prone	40		7.21	76	30.0	27.7	13.9	27.7	22.5	+ 4.9	96
						Ether/Oxygen	175		Prone	75	25	7.09	96	31.0	28.1	13.9	27.7	19.5	+ 7.9	95
						Ether/Oxygen	235	D	Prone	135	85	7.03	115	33.0	29.5	13.9	27.7	19.3	+ 8.1	100

TABLE I—CONT'D

CASE	AGE	SEX	MEDICATION: MORPH. (MG.)	MEDICATION: SCOP. (MG.)	DIAGNOSIS AND OPERATION	ANESTHESIA: CONDITIONS	ANESTHESIA: TIME (MIN.)	ANESTHESIA: DEPTH	POSITION	(MIN.)	PLEURA OPEN (MIN.)	pH, 38° C.	pCO_2 (MM. HG)	TOTAL CO_2 (MM./L)	(HCO_3^-) (MM./L)	HGB. (GM./100)	β	(HCO_3^-) CORRECTED TO pH 7.4 (MM./L)	CHANGE IN FIXED ACID	BLOOD O_2 SAT. (%)
8	75	M	5.4	0.432	Carcinoma	Control						7.36	46	25.3	23.9	13.6	27.2	22.8		91
						Post-sed.	60		Supine			7.23	62	26.0	24.1	12.4	25.6	19.8	+ 3.0	88
					Pneumonectomy	Post-sed.	105		Supine			7.33	48	25.0	23.6	12.4	25.6	21.8	+ 1.0	89
						Oxygen			Prone	10		7.20	62	25.0	23.1	12.4	25.6	18.0	+ 4.8	93
						Oxygen			Prone	60	35	7.13	70	23.4	21.3	12.4	25.6	14.4	+ 8.4	97
						Oxygen		L	Prone	120	95	7.21	60	27.6	25.8	12.4	25.6	21.0	+ 1.8	90
9	20	M	11.0	0.432	Tuberculosis	Post-sed.	75		Supine			7.34	50	26.4	24.9	16.7	31.6	23.0		96
						Ether/Oxygen	49		Prone	54		7.05	74	21.8	19.6	16.5	31.3	8.7	+14.3	99
					Lobectomy	Ether/Oxygen	100		Prone	105	50	6.99	86	22.7	20.1	15.7	30.3	7.7	+15.3	98
						Ether/Oxygen	178	D	Prone	183	128	7.02	83	21.6	19.1	16.6	31.4	7.2	+15.8	93
10	36	F	50.0 (Dem.)	0.324	Tuberculosis	Post-sed.	75		Supine			7.28	52	23.9	22.3	13.7	27.5	19.0		86
						Control			Prone	10		7.37	41	24.6	23.4	13.7	27.5	22.6	− 3.6	93
					Lobectomy	Ether/Oxygen	35		Prone	60		7.45	35	24.2	23.2	13.7	27.5	24.5	+ 5.5	92
						Ether/Oxygen	65	L	Prone	90	25	7.28	50	23.6	22.1	13.7	27.5	18.8	+ 0.2	88
11	43	M	11.0	0.432	Bronchiectasis	Post-sed.	45		Supine			7.39	46	27.4	26.0	14.5	28.5	25.7		91
						Ether/Oxygen	50		Prone	50		7.15	70	24.3	22.2	14.6	28.6	15.1	+10.6	94
					Lobectomy	Ether/Oxygen	103		Prone	103	15	7.27	64	28.8	26.9	15.2	29.6	23.1	+ 2.6	84
						Ether/Oxygen	210	D	Prone	210	122	7.07	97	30.3	27.4	15.1	29.3	17.7	+ 8.0	98
						Control			Supine			7.31	52	26.0	24.4	14.6	28.6	21.8	+ 3.9	80
12	43	F	8.1	0.324	Bronchiectasis	Post-sed.	55		Supine			7.42	40	25.7	24.5	12.0	25.1	25.0		95
						Ether/Oxygen	50		Prone	30		7.25	72	25.2	23.0	12.5	25.8	19.1	+ 5.9	95
					Lobectomy	Ether/Oxygen	95		Prone	75	32	7.23	60	24.8	23.0	12.2	25.5	18.7	+ 6.3	91
						Ether/Oxygen	165	D	Prone	145	102	7.18	66	24.6	22.6	12.9	26.4	16.8	+ 8.2	94
						Control						7.24	57	24.5	22.8	12.4	25.6	18.7	+ 6.3	92
13	44	M	11.0	0.432	Tuberculoma	Post-sed.	75		Supine			7.42	40	25.5	24.3	14.6	28.7	23.7		98
						Ether/Oxygen	40	D	Prone	50	10	7.34	48	25.6	24.2	14.6	28.7	22.5	+ 1.2	
					Thoracotomy	Control			Supine			7.20	62	24.7	22.8	14.2	28.1	17.2	+ 6.5	92

14	51	M	11.0	0.432	Carcinoma	Post-sed.	85		Supine			7.55	22	20.0	19.3	13.7	27.4	23.4		91
						Post-sed.	95		Prone	10		7.61	19	19.7	19.1	13.7	27.4	24.9	− 1.5	95
					Pneumonectomy	Ether/Oxygen	23	L	Prone	63	13	7.37	45	25.6	24.3	13.7	27.4	23.5	− 0.1	96
						Post-op.	225		Supine			7.43	40	26.2	25.0	13.7	27.4	25.8	− 2.4	87
15	54	M	8.1	0.432	Adenoma	Post-sed.	75		Supine			7.40	41	25.0	23.8	14.1	28.0	23.8		92
						Ether/Oxygen	30		Prone	45		7.17	56	20.5	18.8	13.5	27.2	12.6	+11.2	93
					Lobectomy	Ether/Oxygen	50		Prone	65	17	7.38	36	21.3	20.2	13.9	27.8	19.7	+ 4.1	92
						Ether/Oxygen	122		Prone	137	89	7.24	52	22.1	20.5	13.5	27.2	16.2	+ 7.6	91
						Control			Supine			7.38	37	21.8	20.7	12.9	26.4	20.2		91
16	73	F	4.0	0.324	Bronchiectasis	Post-sed.	85		**Supine**			7.50	26	20.5	19.7	12.1	25.3	22.2		90
						Control			Prone	10		7.52	21	17.7	17.1	12.1	25.3	20.1	+ 2.1	90
					Lobectomy	Ether/Oxygen	22		Prone	37		7.42	37	24.0	22.9	12.1	25.3	24.9	− 2.7	95
						Ether/Oxygen	35	L	Prone	50	10	7.21	56	22.8	21.1	12.1	25.3	16.3	+ 5.9	87
17	66	M	35.0 (Dem.)	0.324	Carcinoma	Post-sed.	60		Supine			7.30	51	25.1	23.6	12.5	25.8	21.0		94
						Ether/Oxygen	36		Prone	51		7.38	47	27.0	25.6	11.8	24.8	25.1	− 4.1	98
					Thoracotomy	Ether/Oxygen	72		Prone	87	32	7.35	47	25.5	24.1	12.8	26.3	22.8	− 1.8	96
						Ether/Oxygen	132	L	Prone	147	92	7.48	33	23.8	22.9	13.0	26.5	25.0	− 4.0	97
						Control			Supine			7.51	29	23.0	22.1	13.0	26.5	25.0	− 4.0	84
18	70	F	50.0 (Dem.)	0.324	Bronchiectasis	Post-sed.	80		Supine			7.34	52	27.6	26.0	12.6	25.8	24.5		95
						Ether/Oxygen	52		Prone	12		7.33	50	25.9	24.4	13.9	27.8	22.5	+ 2.0	96
					Lobectomy	Ether/Oxygen	85	D	Prone	45	14	7.04	89	26.0	23.3	13.5	27.0	13.6	+10.9	92
						Control			Supine			7.16	75	26.5	24.2	12.5	25.8	18.0	+ 6.5	88
19	67	M	25.0 (Dem.)	0.324	Carcinoma	Ether/Oxygen	10		Supine			7.17	78	28.5	26.2	10.6	23.1	20.9		91
						Ether/Oxygen	47		Prone	17		7.17	78	28.0	25.7	10.6	23.1	20.4	+ 0.5	89
					Pneumonectomy	Ether/Oxygen	76	D	Prone	46	16	7.02	114	29.6	26.2	10.6	23.1	17.3	+ 3.6	78
20	59	M	8.1	0.324	Bronchial fistula	Ether/Oxygen	9		Prone	54		7.33	45	23.4	22.1	14.5	28.6	20.1		95
					Thoracotomy	Ether/Oxygen	33	D	Prone	78	18	7.09	86	27.8	25.2	14.5	28.6	16.3	+ 3.8	
21	55	M	8.1	0.324	Carcinoma	Ether/Oxygen	20		Prone			7.21	43	17.8	16.5	10.8	23.5	12.1		
						Ether/Oxygen	53	L	Prone		28	7.31	50	25.0	23.5	14.5	28.5	20.9	− 8.8	89
					Thoracotomy	Control						7.39	39	23.3	22.1	15.3	29.7	21.8	− 9.7	86
22	43	F	8.1	0.432	Bronchiectasis	Ether/Oxygen	45		Prone	65		7.23	43	18.5	17.2	13.2	26.8	12.7		99
						Ether/Oxygen	95		Prone	115	45	7.37	32	19.0	18.0	13.4	27.1	17.2	− 4.5	86
					Lobectomy	Ether/Oxygen	115		Prone	135	65									
						Ether/Oxygen	120	L	Prone	140	70	7.36	35	20.4	19.4	13.8	27.6	18.3	− 5.6	83
23	60	M	8.1	0.324	Carcinoma	Ether/Oxygen	50		Prone	55		7.07	92	28.7	25.9	12.8	26.2	17.3		98
					Pneumonectomy	Ether/Oxygen	110	D	Prone	115	35	7.31	57	28.4	26.7	12.8	26.2	24.4	− 7.4	83

TABLE II. EFFECT OF SEDATION

CASE	BEFORE AGE	BEFORE pH_s 38° C.	BEFORE pCO_2 (MM. HG)	BEFORE TOTAL CO_2 (mM/L.)	BEFORE O_2 SAT. %	SECOND SEDATION M.S. (MG.)	SECOND SEDATION SCOP. (MG.)	AFTER pH_s 38° C.	AFTER pCO_2 (MM. HG)	AFTER TOTAL CO_2 (mM/L.)	AFTER O_2 SAT. %
1	28	7.45	28	19.8	89	8.1	0.324	7.19	50	19.5	89
2	28	7.42	34	22.0	97	11.0	0.432	7.21	52	21.4	95
3	48	7.43	41	27.1	92	11.0	0.432	7.31	50	24.7	87
4	48	7.39	43	25.6	99	11.0	0.432	7.31	54	27.0	96
5	50	7.30	47	22.9	90	11.0	0.432	7.21	52	21.3	93
6	56	7.35	42	23.5	89	8.1	0.324	7.31	44	22.3	93
7	63	7.41	45	28.5	92	75.0 (Dem.)	0.324	7.21	66	25.9	90
8	75	7.36	46	25.3	91	5.4		7.23	62	26.0	88
Avg.	49.5	7.39	40.7	24.3	92.4	9.4 (M.S.)	0.385	7.25	53.8	23.5	91.4

there may be a slight decrease in CO_2 tension with an increase in time. A confidence interval, based on all eighteen observations for the average of pCO_2 mm. Hg, is 41.8 = 52.6. These observations do not rule out the possibility that they have come from a series in which the normal value for pCO_2 is 44 mm. Hg.

The significance of this evaluation is that the depressant effects of sedation on respiration are not predictable in patients with pulmonary lesions requiring thoracic surgery. These figures indicate that preoperative sedation with pentobarbital plus morphine and scopolamine can cause a retention of carbon dioxide in some patients. The postsedation values for arterial oxygen saturation were within a normal range.

TABLE III. EFFECT OF SEDATION

CASE	AGE	DIAGNOSIS	SECOND SEDATION M.S. (MG.)	SECOND SEDATION SCOP. (MG.)	TIME AFTER SEDATION (MIN.)	pH_s 38° C.	pCO_2 (MM. HG)	TOTAL CO_2 (mM/L.)	O_2 SAT. %
1	28	Aneurysm lung	8.1	0.324	60	7.19	50	19.5	89
2	28	Tuberculosis	11.0	0.432	60	7.21	52	21.4	95
3	48	Bronchiectasis	11.0	0.432	135	7.31	50	24.7	87
4	48	Lipoma	11.0	0.432	45	7.31	54	27.0	96
5	50	Carcinoma	11.0	0.432	60	7.21	52	21.3	93
6	56	Carcinoma	8.1	0.324	75	7.31	44	22.3	93
7	63	Carcinoma	75.0 (Dem.)	0.324	65	7.21	66	25.9	90
8	75	Carcinoma	5.4		60	7.23	62	26.0	88
9	20	Tuberculosis	11.0	0.432	75	7.34	50	26.4	96
10	36	Granuloma	50.0 (Dem.)	0.324	75	7.28	52	23.9	86
11	43	Bronchiectasis	11.0	0.432	45	7.39	46	27.4	91
12	43	Bronchiectasis	8.1	0.324	55	7.42	40	25.7	95
13	44	Tuberculoma	11.0	0.432	75	7.42	40	25.5	98
14	51	Carcinoma	11.0	0.432	85	7.55	22	20.0	91
15	54	Adenoma	8.1	0.432	75	7.40	41	25.0	92
16	73	Bronchiectasis	4.0	0.324	85	7.50	26	20.5	90
17	66	Carcinoma	35.0 (Dem.)	0.324	60	7.30	51	25.1	94
18	70	Granuloma	50.0 (Dem.)	0.324	80	7.34	52	27.6	95
Avg.	49.8		9.3 (M.S.)	0.360	69.4	7.328	47.2	24.1	92

TABLE IV. PRONE POSITION (OVERHOLT), CHEST CLOSED

ETHER ANESTHESIA, LIGHT							ETHER ANESTHESIA, SURGICAL STAGE						
CASE	AGE	ANESTHESIA (MIN.)	pH_s 38° C.	pCO_2 (MM. HG)	TOTAL CO_2 (MM/L.)	O_2 SAT. %	CASE	AGE	ANESTHESIA (MIN.)	pH_s 38° C.	pCO_2 (MM. HG)	TOTAL CO_2 (MM/L.)	O_2 SAT. %
2	28	40	7.43	30	20.0	98	3	48	70	7.30	56	27.0	92
4	48	15	7.33	54	28.4	100	6	56	85	7.35	37	20.6	93
8	75	60	7.20	62	25.0	93	7	63	140	7.21	76	30.0	96
10	36	35	7.45	35	24.2	92	9	20	49	7.05	74	21.8	99
15	54	30	7.17	56	20.5	93	11	43	50	7.15	70	24.3	94
16	73	22	7.42	37	24.0	95	12	43	50	7.25	72	25.2	95
17	66	36	7.38	47	27.0	98	18	70	52	7.33	50	25.9	96
22	43	45	7.23	43	18.5	100	19	67	47	7.17	78	28.0	89
							20	59	9	7.33	45	23.4	95
							23	60	50	7.07	92	28.7	98
Avg.	53	35	7.33	45.5	23.5	96		52.9	60	7.22	65.0	25.5	94.7

Table IV shows the effect of the light and moderate surgical states of ether anesthesia on the arterial blood and pH_s, carbon dioxide tension, and oxygen saturation, before opening the pleura, with the patient in the prone position. Seven of the ten patients who were in the moderate surgical state of anesthesia had respiratory acidosis; in Case 23, there was an elevated pCO_2 (92 mm. Hg) and a pH of 7.07—a severe respiratory acidosis. In the group of patients who were lightly anesthetized, the highest pCO_2 was 62 mm. Hg, with a pH of 7.20.

Increasing the depth of ether anesthesia, with the patient in the prone position, evidently depresses effective alveolar ventilation as shown by a retention of carbon dioxide.

Analyses of the effects of lobectomy, pneumonectomy, and light and moderate surgical states of ether anesthesia on the excretion of carbon dioxide are shown in Tables V and VI. The observed difference between the average pCO_2

TABLE V. PRONE POSITION (OVERHOLT), CHEST OPEN

LOBECTOMIES							PNEUMONECTOMIES						
CASE	AGE	CHEST OPEN (MIN.)	pH_s 38° C.	pCO_2 (MM. HG)	TOTAL CO_2 (MM/L.)	O_2 SAT. %	CASE	AGE	CHEST OPEN (MIN.)	pH_s 38° C.	pCO_2 (MM. HG)	TOTAL CO_2 (MM/L.)	O_2 SAT. %
1	28	35	7.00	82	20.6	81	3	48	55	7.08	109	34.1	79
9	20	50	6.99	86	22.7	98	5	50	105	7.14	72	24.3	90
10	36	25	7.28	50	23.6	88	6	56	40	7.10	76	23.6	96
11	43	122	7.07	97	30.3	98	7	63	85	7.03	115	33.0	100
12	43	105	7.18	66	24.6	94	8	75	95	7.21	60	27.6	90
15	54	90	7.24	52	22.1	91	14	51	13	7.37	45	25.6	96
16	73	10	7.21	56	22.8	87	19	67	16	7.02	114	29.6	78
18	70	14	7.04	89	23.3	92	23	60	35	7.31	57	28.4	83
22	43	70	7.36	35	20.4	83							
Avg.	45.5	58	7.15	68	23.4	90		59	56	7.16	81	28.3	89

TABLE VI. PRONE POSITION (OVERHOLT), CHEST OPEN

ETHER ANESTHESIA, LIGHT								ETHER ANESTHESIA, SURGICAL STAGE							
CASE	AGE	ANESTHESIA (MIN.)	CHEST OPEN (MIN.)	pH, 38° C.	pCO_2 (MM. HG)	TOTAL CO_2 (MM/L.)	O_2 SAT. %	CASE	AGE	ANESTHESIA (MIN.)	CHEST OPEN (MIN.)	pH, 38° C.	pCO_2 (MM. HG)	TOTAL CO_2 (MM/L.)	O_2 SAT. %
4	48	60	5	7.36	43	24.2	100	1	28	105	35	7.00	82	20.6	81
8	75	120	95	7.21	60	27.6	90	3	48	135	55	7.08	109	34.1	79
10	36	65	25	7.28	50	23.6	88	5	50	150	105	7.14	72	24.3	90
13	44	40	10	7.34	48	25.6	—	6	56	130	40	7.10	76	23.6	96
14	51	23	13	7.37	45	25.6	96	7	63	235	85	7.03	115	33.0	100
15	54	137	89	7.24	52	22.1	91	9	20	178	128	7.02	83	21.6	93
16	73	35	10	7.21	56	22.8	87	11	43	210	122	7.07	97	30.3	98
17	66	127	92	7.48	33	23.8	97	12	43	165	102	7.18	66	24.6	94
22	43	120	70	7.36	35	20.4	83	19	67	76	16	7.02	114	29.6	78
								20	59	33	18	7.09	86	27.8	—
								23	60	110	35	7.31	57	28.4	83
Avg.	54	81	45	7.32	47	23.9	91.5		49	139	67	7.09	87	27.1	89

of 68 mm. Hg and pH of 7.15 for lobectomies, and the pCO_2 of 81 mm. Hg and pH of 7.16 for pneumonectomies, with pleura open, may well be due to mere sampling fluctuations. The observed difference between the average pCO_2 of 47 mm. Hg and pH of 7.32 for light anesthesia, and the average pCO_2 of 87 mm. Hg and pH of 7.09 for the moderate surgical state of anesthesia, with the pleura open, is highly significant. The variability of pCO_2 values was investigated by a two-way analysis of variance, based on the type of operative procedure and the depth of anesthesia. Table VII is an analysis of the variance.

TABLE VII

SOURCE OF VARIATION	SUM OF SQUARES	D.F.	MEAN SQUARE	F	TABULATED 5%	F 1%
Operation (lobectomy, pneumonectomy)	131.56	1	131.56	0.4		
Anesthesia (light, moderate)	4998.56	1	4998.56	16.0	4.67	9.07
Interaction	7.75	1	7.75			
Individuals	4061.	13	312.4			

The F value for depth of anesthesia is highly significant, whereas the F value for operation is not significant. The T test for the difference of pCO_2 between the light and moderate states of anesthesia gives a value of T = 2.76, with 16 degrees of freedom. This is significant at the .05 level.

These figures are evidence that the decrease in excretion of carbon dioxide (the state of respiratory acidosis) is as likely to occur during the procedure of a lobectomy as it is during a pneumonectomy. The analysis of variance demonstrates that during intrathoracic surgery in the prone position, the more significant factor in causing respiratory acidosis is the depth of anesthesia, and not the type of pulmonary resection. From Table VIII it can be seen that there was no significant change in the carbon dioxide tension after the patients were turned from the supine to the prone position.

TABLE VIII. pH AND pCO_2 AFTER ENDOTRACHEAL INTUBATION

EFFECT OF SUPINE AND PRONE POSITIONS BEFORE ANESTHESIA				
	SUPINE		PRONE	
10 PATIENTS	pH_s 38° C.	pCO_2 (MM. HG)	pH_s 38° C.	pCO_2 (MM. HG)
Average	7.41	36	7.41	32
EFFECT OF SUPINE AND PRONE POSITIONS DURING ETHER ANESTHESIA				
	SUPINE		PRONE	
16 PATIENTS	pH_s 38° C.	pCO_2 (MM. HG)	pH_s 38° C.	pCO_2 (MM. HG)
Average	7.24	64	7.27	58

Further analysis does not indicate any relation between the length of time the pleura is open and the excretion of carbon dioxide. The course of the CO_2 tension in eight representative intrathoracic procedures is shown in Fig. 1, from which it is apparent that the length of time the pleura remains open has no bearing on the CO_2 tension. Fig. 2 shows the lack of correlation between the length of time the pleura remains open and CO_2 tension during twenty-three intrathoracic procedures.

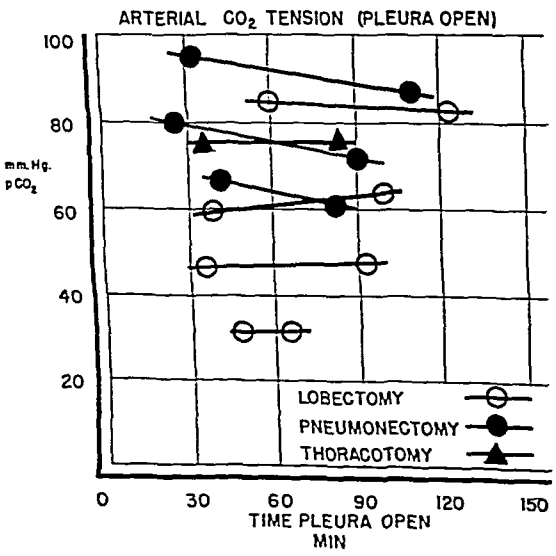


Fig. 1.

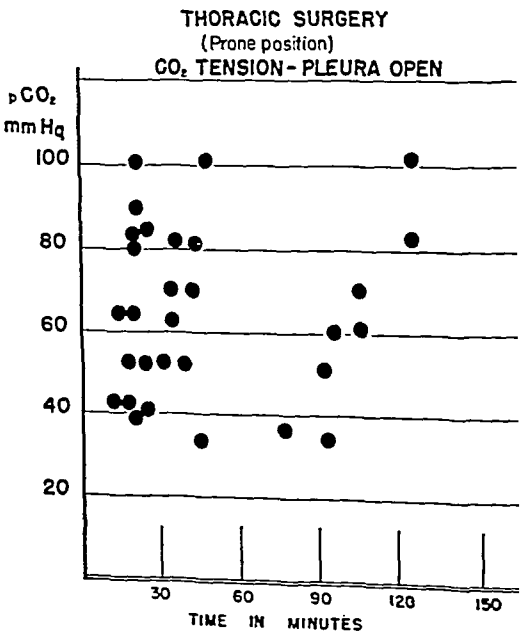


Fig. 2.

Fig. 1.—The figure shows that time had no effect on the arterial pCO_2 while the pleura was open (eight representative intrathoracic procedures).

Fig. 2.—The arterial blood CO_2 tension is plotted against the time the pleura was open (thirty intrathoracic procedures). The figure indicates the lack of correlation between these two factors.

Table IX shows that in twenty-three cases, the average of the fixed acids during ether anesthesia for lobectomy or pneumonectomy was within a normal range of variation (−0.8 to +4.4 mM per liter). In several patients the fixed

TABLE IX. ACID-BASE EQUILIBRIUM, PRONE POSITION

	pH_a 38° C.	pCO_2 (MM. HG)	TOTAL CO_2 (MM/L.)	(HCO_3^-) (MM/L.)	HGB. (GM./ 100)	β	(HCO_3^-) CORRECTED TO pH 7.4	FIXED ACID (MM/L.)
Ether Anesthesia, Light								
Preoperative (supine)	7.36	44.4	23.8	22.8	13.5	27.1	21.2	
Chest closed	7.33	45.5	23.5	22.1	12.8	26.2	20.3	−0.8
Chest open	7.32	47.0	23.9	22.6	13.5	27.2	20.4	−0.9
Ether Anesthesia, Surgical Stage								
Preoperative (supine)	7.30	50.0	24.5	23.0	13.5	25.0	20.4	
Chest closed	7.22	65.0	25.5	23.5	13.5	27.2	18.7	−1.7
Chest open	7.09	87.0	27.1	24.5	13.7	27.4	16.0	−4.4

acids were elevated. This occurred in a few patients who had slight elevations of CO_2 tensions. In other instances, where the CO_2 tensions were extremely high, the fixed acids were not elevated.

DISCUSSION

The results of this study show that the Overholt prone position does not aid in the excretion of carbon dioxide during thoracic surgery. Preoperative sedation, depth of ether anesthesia, and an open pneumothorax are the principal factors that may cause a retention of carbon dioxide tension in the arterial blood. It is recognized that there are other factors in addition to these that may produce an increase in alveolar carbon dioxide tension and a fall in pH during thoracic surgery. Among these are ligation of a bronchus previous to ligation of the pulmonary vessels, an open bronchus during lobectomy or pneumonectomy, circulation of blood through inadequately ventilated pulmonary tissue, impairment of pulmonary and systemic circulation, and diminished blood flow through the lung. When these causes are corrected, the carbon dioxide tension is maintained at the pre-existing level, dependent on the depth of anesthesia.

The arterial carbon dioxide tension is considered to equal the mean of the entire range of alveolar carbon dioxide pressure existing in different parts of the lung and at different movements in the ventilatory cycle.[12] The increase in arterial pCO_2 during intrathoracic surgery can be attributed to decreased alveolar ventilation and to the mixing of blood that has passed through inadequately ventilated alveoli with blood that has passed through the well-ventilated lung. The face-down position is not an aid to the ventilation of the lung exposed to atmospheric pressure during the course of thoracic surgery. The exposed lung can be considered functionless; it acts only as a reservoir for venous blood having a high carbon dioxide tension. When pneumonectomy or lobectomy is performed, the increase in carbon dioxide tension is most likely due to a deficiency in the respiratory ventilating mechanisms.

The opening of the pleura impairs the bellows action of the muscles of respiration. Respiratory studies[5] demonstrate that an open pleura (see Fig. 3) reduces the effective tidal volume and alveolar ventilation. Respirations

decrease in amplitude but increase in rate. The resultant increase in respiratory minute volume does not, however, improve the effective tidal exchange. Although the prone position favors an increase in diaphragmatic excursions, the increase is not sufficient to aid the excretion of carbon dioxide.

The respiratory-stimulant action of ether and the reflex surgical stimulation in the light stage of anesthesia can cause an increase in effective tidal volume and respiratory minute volume. In our series, the group that was lightly anesthetized with ether had respiratory rates ranging from 40 to 50 per minute during surgery. Whether a respiratory rate of this magnitude is physiologically sound is questionable because of the increased work of breathing and exaggerated excursions of the muscle wall, diaphragm, and mediastinum. Surely, excessive respiratory movements must impede a meticulous surgical dissection within the thorax.

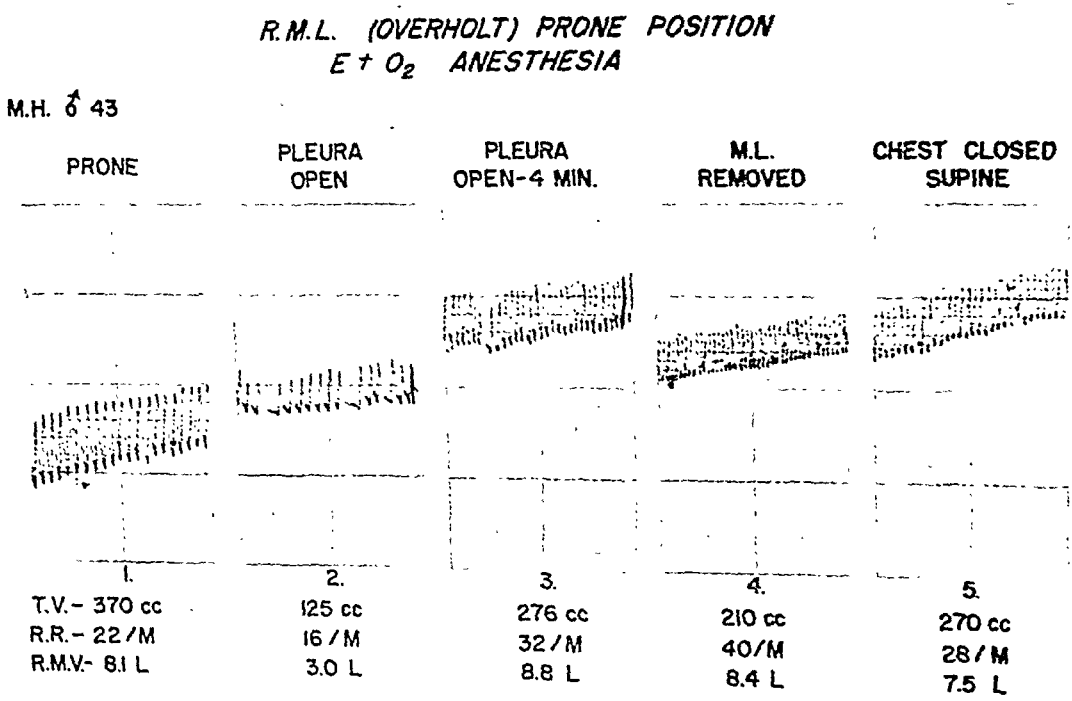


Fig. 3.—Spirogram shows the decrease in effective ventilation during a lobectomy in the prone position.

Comparison of the arterial blood carbon dioxide levels in the group of patients lightly anesthetized and in the group in the midplane of surgical anesthesia demonstrates that it is the depth of ether anesthesia, and not the type of pulmonary resection, that decreases the excretion of carbon dioxide. It can be postulated that in the moderate state of surgical anesthesia the threshold of the respiratory center is elevated and does not respond to reflex surgical stimuli. It is evident that a severe respiratory acidosis may occur during

lobectomy as well as during pneumonectomy. The *effective* respiratory minute volume and *effective* alveolar ventilation are not increased during the surgical state of ether anesthesia when the chest is open. Serial blood gas determinations taken when the pleura was open indicate that the carbon dioxide retention does not increase with time. Analyses of these values are shown in Figs. 1 and 2. Since the alveolar carbon dioxide tension is inversely proportional to alveolar ventilation,[11] it follows that the factors that impair alveolar ventilation are responsible for an elevated pCO_2. If the level of anesthesia is maintained throughout, then the time the pleura is open need not be considered. It is recognized that frequent arterial blood determinations are necessary in order to plot the complete course of the alveolar pCO_2 and correlate it with the etiological factors. A single determination in a single patient, taken when the pleura is open, does not permit a true evaluation. Neither does a comparison of the effect of time in a group of patients, based on single determinations, show any positive correlation.

In this study, the arterial oxygen saturation remained within normal limits after preoperative sedation. This finding is in contrast to a reported[6] average of 88 per cent oxygen saturation of arterial blood after sedation in patients for thoracic surgery; it may be that large doses of depressant drugs were used for premedication, or blood determinations were taken at the height of the depressant effects of the drugs.

The arterial blood oxygen saturation was within a normal range in all but two patients during surgery. The patient in Case 1 had a pulmonary arteriovenous aneurysm, and the respired high oxygen atmosphere did not alter the preoperative arterial oxygen saturation level. The prone position had no influence on the arterial oxygen saturation, in either the nonanesthetized or anesthetized state. Sixteen minutes after the pleura was opened the patient in Case 7 had an arterial oxygen saturation of 78 per cent, pH of 7.02, and pCO_2 of 114 mm. Hg. The lowered arterial oxygen saturation was most likely due to a decrease in alveolar ventilation precipitated by the opening of the pleura.

SUMMARY AND CONCLUSIONS

This has been an evaluation of the etiological factors in respiratory acidosis during thoracic surgery. It has been demonstrated that the prone (Overholt) position does not prevent an increase in carbon dioxide tension during either lobectomy or pneumonectomy.

Dangerously high levels of carbon dioxide tension were encountered when the hemithorax was open and the patients allowed to breathe spontaneously during ether anesthesia. A lowering of the pH and increase in carbon dioxide tension occurred during the surgical state of ether anesthesia, with the patients in the prone position.

Analysis of the effect of lobectomy, pneumonectomy, depth of ether anesthesia, and duration of operation reveals the following: (1) An increase in carbon dioxide tension may occur with the same frequency and severity during

lobectomy as during pneumonectomy; and (2) the severity of the respiratory acidosis is not a factor of time but is a factor of the depth of ether anesthesia during intrathoracic surgery.

Statistical analysis was made by Dr. Gottfried E. Noether, Associate Professor of Mathematics (Statistical Division), Boston University College of Liberal Arts, Boston.

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A MODIFIED THORACOTOMY TECHNIQUE FOR THE PREVENTION OF POSTOPERATIVE PAIN AND DEFORMITY

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OPERATIONS upon the thoracic cavity may be divided into two groups: those which require collapse of the chest wall, and those which do not. Collapse may be considered necessary in the resectional treatment of pulmonary tuberculosis or to prevent mediastinal shift when the hemithorax is left empty. Otherwise, the resection of ribs is generally done to obtain exposure. In our work, we have felt that removal of a rib increases the difficulty of obtaining airtight closure and often results in a groove in the chest wall along the axis of the resected rib. The importance of this feature was brought forcibly to our attention when a young woman threatened a malpractice suit for a bad cosmetic result, although she admitted that the operation had cured her of bronchiectasis.

In addition, we have been led by our results to believe that, where there is a poor alignment of sectioned ribs and/or excessive movements of the anterior segments, a large callous formation may ensue. If the posterior end of the intercostal nerve becomes entangled in this callus, it may cause the late development of disabling pain. Four of our patients required the operative liberation of the nerve trunk for relief of pain six to eighteen months after resection. These operations were only partially successful.

The standard procedure for posterolateral thoracotomy was laid down as long ago as 1925 by Lilienthal.[1] Later work, particularly by Overholt and Langer,[2] and Klassen,[3] shows that the search for improvement is continuous. Unfortunately, some of the refinements which yield an improvement in results are paid for at a cost of increased operating time. In our work, many methods—those described by the foregoing authors, as well as drilling and diagonal cutting, followed by wiring—were tried and abandoned before the simple method to be described was developed.

It seemed rational to assume that chest deformity could be avoided by conserving, as nearly normal as possible, both the substance and position of the ribs. It also seemed logical to expect that postoperative fixation of the ribs in good position would prevent, to a large extent, the production of excessive callus.

It is our opinion that the thoracotomy technique described here accomplishes these purposes. In all illustrations, the patient is in the face-down position.

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TECHNIQUE

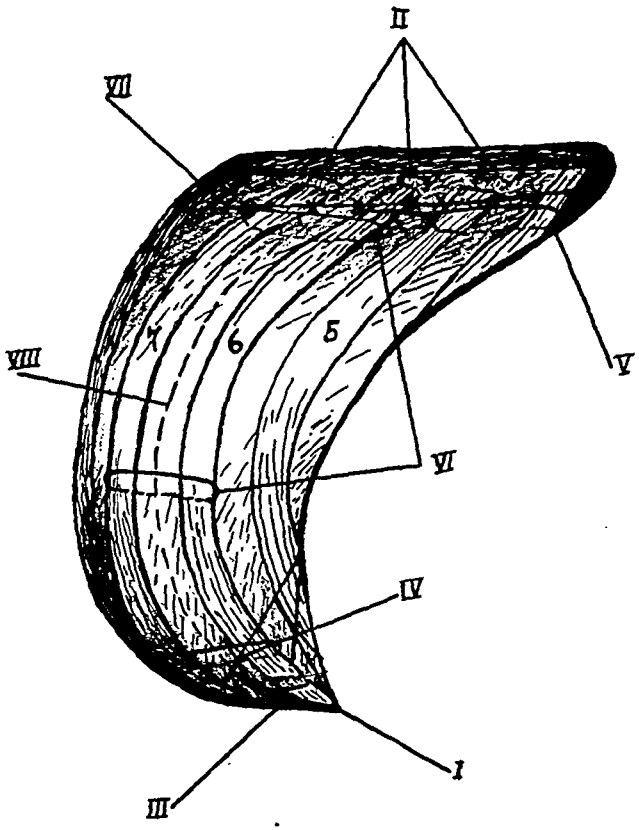
An incision is made, beginning at a point halfway between the fourth dorsal spine and the scapular border, and carried in a curved sweep around the normal position of the angle of the scapula to a point represented by the intersection of the sixth intercostal space and the anterior axillary line. All structures down to the muscles are divided throughout the entire length of this incision. Towels are sutured to the fascia along the margins of the



FIG. 1.—Photograph of anterior angle of wound. The three instruments, from left to right, are between the skin and muscle, between the muscle and ribs, and within the chest cavity at the anterior sectioned end of the sixth rib.

wound. The muscles are then divided from the beginning of the skin incision to a point represented by the intersection of the sixth intercostal space and the midaxillary line. From this point, the interdigitations of the serratus anterior and the external oblique abdominal muscles are not cut. The skin is then dissected away from the external surface of the muscular attachments for a distance of about one inch on either side. The inner surfaces of these muscles

are next freed from their attachments to the sixth and seventh ribs for a distance of about two inches (Fig. 1). In this maneuver, the surgeon should avoid separation of the fused muscle bundles from each other or perforation of the thin musculomembranous wall from the inside. Segments, one centimeter in length, are removed subperiosteally from the fifth, sixth, and seventh



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Fig. 2.—Semidiagrammatic drawing of the closed thorax, showing the following:

I. Anterior end of skin incision.

II. Sectioned fifth, sixth, and seventh ribs.

III. Sectioned anterior end of sixth rib.

IV. Pericostal suture incorporating interdigitations of anterior serratus and external abdominal oblique muscles (shown in outline).

V. Suspension suture from fourth rib supporting free ends of fifth, sixth and seventh ribs.

VI. Pericostal sutures to approximate intercostal muscles in sixth interspace.

VII. Aponeurosis of iliocostalis muscle (outline) tacked down by interrupted sutures to intercostal muscles.

VIII. Intercostal suture line. *This becomes airtight without sutures.*

The drawing shows the right side of the chest, with the patient in the face-down position.

ribs posterior to the angle. Care must be taken not to strip away more periosteum than is essential and not to open the pleura prematurely.

The thoracic cavity is now opened by an incision through the midline of the sixth intercostal space. It is very important to keep the line of incision halfway between the two ribs. This is best accomplished by spreading the posterior free ends of the ribs by means of rake retractors to maintain tension on the intercostal structures as the incision advances. The fifth and sixth



Fig. 3.—Reproduction of x-ray picture of left upper lung field three months postoperative. The suspension suture has, apparently, become enveloped in osteoblastic cells. The sixth and seventh ribs are in good position, although the fifth rib is held slightly too high. There is little evidence of callous formation.

intercostal vessels are tied and cut. The corresponding nerves are cut, but not included in the tie. Rib spreaders are then inserted and opened to about four inches. Adequate exposure is provided to approach the sixth rib from the inside and a segment is resected through the parietal pleura opposite the end of the skin incision, taking care to liberate completely the proximal end of the rib from all attachments. This may require an extension of the intercostal incision.

The rib spreader is removed. The floating sixth rib is elevated and placed above the fifth rib. After replacement of the spreader, a good exposure is easily obtained.